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SCLEROSIS OF THE RETINAL VESSELS

BY

N. PINES

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(*Concluded*)

The Retinal Vessels in Sclerosis of the General Vascular System

Syphilis

Syphilis has an immemorial reputation as the greatest of vascular damagers, but it chiefly attacks definite parts of the system and is a good demonstration of the rule that different toxins have their spots of predilection in the patient's body. In the case of the central nervous system, the pathology of syphilis points out again the correctness of Weigert's sentence; being a foreign body to the proper nervous tissue, the vascular system may either be the chief sufferer (*e.g.*, gumma) or may more or less escape when the proper nervous tissue is greatly damaged (tabes dorsalis, general paralysis of the insane). But even in tabes, the aorta is often attacked, a striking difference from the retinal vessels. Pulay, of Vienna, found by X-rays syphilis of the aorta present in 56 per cent. of syphilitics of all ages. Kessler, of Vienna, made a post-mortem of 60 patients, who died from tabes, and in two-thirds of them he found definite syphilitic affection of the

aorta or of the aortic valves; the cause of this mesaortitis (tabetica) is not different in character from the ordinary mesaortitis specifica, although Frost believed that it is less frequent and more benign in tabetics. If it appears to be more benign clinically, this is because the tabetic usually leads a more restricted mode of life, anti-syphilitic treatment is usually commenced earlier, and, perhaps, the subjective sensation of pain and discomfort is not so sharply felt by a tabetic. Pineles, of Vienna, points also to the possibility of syphilis (mesaortitis, aneurysm, incompetence of the aortic valves, syphilis of the coronary vessels) in the case of "rheumatic" pains in the region of the sternum; in his opinion, this kind of syphilis has become much more frequent since the War. Mars still courts Venus! Bruhns thinks that he proved the presence of syphilis of the aorta in 30 per cent. of 200 cases of syphilitics, who were infected 9-30 years previously. Therefore, in his opinion, every sufferer from syphilis ought to be kept under very careful observation and a radiogram of the aorta should be taken frequently. Prof. Schlesinger makes the diagnosis of a syphilitic aorta if he hears a systolic murmur over the aorta along with an accentuated second aortic sound. *The blood pressure is usually normal.* Very often the patients complain of a severe burning pain behind the sternum. A radiogram shows a diffuse dilatation of the aorta, especially of the ascending arch; *Wassermann's reaction is not always positive.* If the openings of the coronary arteries in the aorta are affected we get the clinical picture of angina pectoris, if the aortic valves, insufficiency of the aorta. Stenocardia, aortic regurgitation, without endocarditis in the patient's history, and aortic dilatations, in younger patients, point to syphilis of the aorta, even if Wassermann's reaction be negative. *Nearly every aneurysm of the aorta is of syphilitic origin.* This view of the frequency of syphilis in aortitis and angina is so widespread now that a word of warning was given in Paris last year, when Danzelot in the Société Médicale des Hôpitaux, according to the *Monde Médicale*, appealed to the profession to be much more reserved in this regard and to try to prove syphilis not only by the Wassermann test but by a very careful bodily examination. Gallavardin in his statistics showed that out of 100 cases of angina pectoris, in 40 syphilis could be entirely eliminated as an aetiological factor, 30 were doubtful, in 9 it was probable, and in 21 it was proved. Especially sarcastic was Laubry: "Pain, radiating in the left arm, an extra systole, provoke a state of anxiety, repeated palpitations are uncomfortable, an attack of dyspnoea of asthmatic character, and the talk starts on true or false angina, and it is very seldom that a fine laboratory examination will not reveal a Wassermann reaction slightly positive and the radiogram with its

shadows and mensurations will not relieve the medical man from his last scruples." Be it as it may, syphilis does attack the aorta and coronary arteries as an area of predilection and it does not behave in the same manner towards the retina. I shall quote from my cases only one, where the Wassermann reaction was negative, at first, in spite of definite syphilitic iritis and, later on, two gummata of the iris, but becoming positive a few weeks afterwards. In two other cases where the blood pressure was normal, nothing abnormal could be detected in the retinal vessels. It was the same with the third case, who had aortitis syphilitica with a positive Wassermann reaction and had an attack of mild coronary thrombosis last year. But the retinal picture of another case where syphilitic aortitis is also present, with a positive Wassermann reaction and who has been brought near death's door by a severe attack of coronary thrombosis with resulting permanent cardiac debility is quite different, because, in my opinion, essential hyperpiesis came into play, his blood pressure being 200/100 (it is only 110/80 now). I had a case of general paralysis of the insane successfully treated by malaria. His retina did not present anything characteristic, his blood pressure being normal. Especially remarkable was another case. Three years ago (No. 8 C. in my first paper) his blood pressure was 160-210/110-130. B.E. translucency absent; l.r. dotted: c.w. colour. Small cilio-retinal arteries were tortuous in the right eye, their lumen changed. All the arteries were narrow. The veins in both eyes were crushed by the arteries, but not deflected. In the left eye a vein was crushed and deflected centripetally. White lines accompanied the vessels at the crossings. In September, 1926, he fell unconscious. I found him on the ground in a state of coma and sent him to hospital with a diagnosis of probable cerebral haemorrhage. The Wassermann reaction proved to be positive in the cerebro-spinal fluid and a diagnosis of cerebro-spinal lues was made at the hospital. When discharged from the hospital his blood pressure was 150/90, fundi unchanged (11/1/27).

After months of treatment he did not improve. He was seen by Dr. Gordon Holmes, who made a diagnosis of general paralysis of the insane, and treated him with malaria; the success was a brilliant one. When discharged from the hospital on June 4, 1926, his blood pressure was 200/110, fundi unchanged.

On December 3, 1927, his blood pressure was 230/130. The veins were not only crushed, but deflected by the arteries. There were many retinal haemorrhages and also white lines on the disc. In the left eye the sup. temp. artery and the vein were running close together with white lines between them. The left was more crushed now than before. His blood urea was normal and urea concentration test satisfactory.

A girl aged 20 years complained of severe headache. Her doctor gave her a certificate to consult an ophthalmic surgeon. Her approved Society sent her to an optician who provided her with glasses. She was not relieved, because she had a double papillitis specifica, with a positive Wassermann reaction. For a few weeks, in her left eye, all the lower retinal vessels were quite clear, but behind them, especially with the increased light of the ophthalmoscope, was seen a white veil on the retina, which accompanied the vessels as a white tract. It disappeared later on. It was, in my opinion, an oedema of the external layers of the retina, probably caused by choroiditis.

Conclusions.—Syphilis *per se* does not especially attack the retinal vessels, but if arterial hypertension is also present the sclerotic changes are well marked and advanced, as in similar cases of essential hyperpiesis.

Atheroma

This serious disease is usually more or less localised. It more often attacks the large arteries, and is especially dangerous when situated in the coronary arteries or just at the point where they leave the aorta, causing either true angina pectoris or a myocardial infarction (coronary thrombosis). But, on the other hand, such an authority as Winkelbach, in a post-graduate lecture, delivered in Vienna in 1924, stated that sclerotic coronary arteries were detected in persons who never suffered from angina pectoris, and that people died from angina whose coronary arteries were not at all sclerotic. Pal also maintains the view that true angina pectoris is often an angiospastic neurosis of the coronary arteries. The pain is caused by an arterial stasis, especially at the point of entering the aorta; the flow of blood is interfered with; the vessels become tortuous and rigid. Sternberg, also of Vienna, quotes Ortner, in his description of abdominal angina, which is characterised by excruciating pain, motor insufficiency of the bowels, sometimes even collapse and a feeling of impending death. The cause of this attack, according to most authorities, is also an angiospastic condition of the intestinal vessels (vascular crisis of Pal). Ryle states that angina "is frequently, but not invariably, associated with changes in the arteries, and particularly—though not necessarily—with atheroma of the first part of the aorta and the coronary vessels." And, if my memory does not fail me, Nassau characterised the narrative of a neuropath, who thinks he has an angina, in a simple, but clever sentence: "There is much more noise than real danger." The true sufferer who has been near death, describes his sensations in a tragic,

brief and precise way, as opposed to the long, fantastic and full of " fine feelings " story of the neurotic.

It is different with coronary thrombosis. Here there is always atheroma present. The final blow to the diseased vessel is thrombosis (Parkinson and Bedford). This pathological condition differs from angina in the prolonged time of the attack, vomiting, collapse, and sometimes tenderness in the epigastrium, which may lead to a diagnosis of acute abdominal trouble. Winkelbach says that if the patient survives a severe attack of coronary thrombosis, he is usually a broken man and can never recover the same vigour of the heart's action as he enjoyed in the past.

What is the state of the retinal vessels in those tragic cases? Foster Moore states definitely in his " Medical Ophthalmology " when speaking of atheroma: " There was no evidence of any considerable disease of the retinal arteries in any case." This is a perfectly correct statement. In two cases, the first having had one of the most dreadful attacks of coronary thrombosis that I have ever witnessed, there is nothing exceptional in the retinae, the blood pressure being normal. Two other cases had definite signs of marked vascular sclerosis of the retina, but I never took their blood pressure before the attack, whilst during the attack and afterwards it may be low because of the myocardial insufficiency. One of them was a powerfully built ex-service man, an ardent follower of Bacchus, who has drunk a bottle of whisky a day regularly for the last few years. I would suggest that there is a slight possibility of differentiating between a false and a true attack of angina. The slightest degree of retinal sclerosis in combination with the subjective symptoms of the patient makes the correct diagnosis more probable; on the other hand, true angina may give a retinal picture of a normal person of corresponding age; the same applies to the heart, where coronary sclerosis may be absent. With arterial hypertension, however, the retinal picture changes immediately. A person with an extreme degree of coronary or aortic atheroma will not present anything specially abnormal in his retinal vessels if the blood pressure be normal; *arterial hypertension, super-added to atheroma, will give a characteristic appearance of marked and often advanced retinal vascular sclerosis.* It may be argued that atheroma here is the result of essential hyperpiesis which existed previously. I should only welcome this criticism, as it entirely coincides with my view, especially for cerebral atheroma. Especially noteworthy and demonstrative of my theory of a special affliction of the retinal vessels in cases of essential hyperpiesis is the following case. It is that of a man aged 59 years, ex-seaman; tall and powerfully built. On November 15, 1926, his blood pressure was 150/80. In his retina the translucency of the vessels was diminished, but still present. No

white lines on the disc or crossings. One vein was deflected centrifugally, but not depressed by the artery (a congenital deflection). On June 10, 1927, the fundi were the same. On September 29, 1927, he came to see me with blood pressure 180/100. Extra systoles; severe retrosternal pain. Fundi: translucency lost, copper-wire colour, light reflex dotted, white lines on the disc and crossings. In the right eye the sup. temp. vein was once deflected centrifugally by the artery whilst the second crossing did not as yet affect it. In the left eye the sup. temp. vein was crossed three times by the artery being not affected by the first, centripetally deflected by the second, and not affected by the third. He was able to walk home. On September 30, 1927, he developed a picture of coronary thrombosis and I sent him with this diagnosis to the London Hospital, without having time to describe his previous history. On October 23, 1927, the House Physician kindly informed me that the man actually had coronary thrombosis and auricular fibrillation as well. "His Wassermann reaction is, however, negative, X-rays show some general dilatation of the aorta, but no aneurysm. I think he must be a case of atheromatous arterial disease and *probably* (italics of the House Physician) had a high blood pressure before this attack." A correct surmise, as is shown by my history of the case. We have previously seen that atheroma with a normal blood pressure does not change the retinal vessels much. My patient had a normal blood pressure, without syphilis in his past, and his fundi were perfectly normal. Enter essential hyperpiesis, and it produces in a short time a complete change in the fundi and coronary atheroma as well. He died from cardiac failure in July, 1928.

Atheroma may attack the eye, too, but here again, it is a localised condition. It means a thrombosis of the vessels, usually in one eye only. In the purely classical, so to speak, form the blood pressure is normal. On the other hand, atheroma in my opinion, is often a complication and result of essential hyperpiesis. A good illustration in the first group is the noteworthy history of the following case. He is a man aged 50 years, who came to see me on August 27, 1926. His blood pressure was 140/100. Arterio-sclerosis of first or second degree of brachial and radial arteries. Under medication his blood pressure came down to 140/90. Fundi: B.E. myopia of 8.0 D.; posterior choroiditis. Arteries were narrow, their colour and light reflex stronger than usual, but not decidedly pathological. The veins were larger. They were not crossed by the arteries, so that I could not define their translucency. On February 13, 1927, his blood pressure was 120/80. He complained of a funny sensation in his right hand. A definite paraesthesia and weakness of the right palm followed four days later. I advised him to consult a nerve specialist and,

frightened, he disappeared. He appeared again on April 12, 1927, with a severe difficulty in his speech, lasting a fortnight or so, and a fresh embolism (thrombosis) of the central artery of his right eye, with complete blindness. The strange part in this remarkable picture was the pupil; enlarged to its maximum, it did not react to light or accommodation, nor consensually with the other pupil. The diagnosis was between arteritis obliterans of a very unusual localisation and neoplasm. A radiogram showed a normal skull. On July 2, 1927, the power of his hand was restored, the speech was better, blood pressure was 125/80. Atrophia n. optici dextri; the pupil did not react to light, but reacted consensually with the other pupil. Now, more than 18 months after the attack, he is in comparatively good health. The other eye has not been affected at all. A diagnosis of neoplasm may now be excluded. His heart is good and he has never shown signs of atheroma anywhere else in his body.

A patient was first seen on January 24, 1926, a man aged 64 years, with a blood pressure of 210/110, urine, sp. gr. 1010, traces of albumen. Fundi: translucency of arteries lost, copper-wire colour, light reflex dotted. White lines at the crossings. The veins were depressed or slightly crushed, without interference with the blood circulation. Venous pulse. Seen again on February 3, 1926, and February 9, 1926, the blood pressure being 210/105. On November 13, 1927, he came to see me complaining that for the last two weeks he had seen on four occasions " a fog " before his right eye; blood pressure 240/120. Fundi: strikingly small changes at the crossings. No silver-wire arteries seen. In the right eye a striking change in the lumen of two independent vessels, an artery and a vein, which were running towards the macula. An irregular haemorrhage near the other independent vessel. In the left eye were many retinal haemorrhages. Striking change in the lumen of many arteries, especially of the sup. temp. art. The sup. vein was deflected centripetally near the disc by a small arterial twig. Blood urea was 51 mgm. per 100 c.c. On December 1, 1927, the blood pressure was 250/125. Urine: sp. gr., 1015, albumen, traces. B.E. as before. On December 3, 1927, he developed thrombosis of the upper branch of the right central artery. All the upper part of the retina was white, oedematous, *but the oedema did not cover the vessels*. Roughly it corresponded to the upper hemisphere. The lower hemisphere was normal in colour. The veins of the upper hemisphere were not affected. The arteries were narrow, covered with a white sheath, but the blood stream was not interrupted. One independent artery, going upwards from the disc, was intact near the disc. Thence it was covered with a white sheath until it divided into two branches. The left branch, after being crossed by a vein, was again covered with a

white sheath. The right branch was intact. In red-free light the border line between the upper and lower hemispheres was beautifully seen; it was a festoon-like line leaving the macula intact. Complete blindness of the lower part of the visual field. Central vision 4/6. He came to see me immediately after he lost his sight, so that the picture was fresh. On December 11, 1927, he felt generally quite all right; very active. The blood pressure was 250/120, in spite of medication, low diet and three days' rest in bed. The main trunk of the arteria temporalis superior dextra was now free from the white sheath. It was only accompanied by white lines on either side. The left branch of the independent artery was still the same. Another artery, which was crossed by a branch of the sup. temp. vein was completely obliterated, the blood-column being interrupted. All other arteries of the affected area were narrow, but not obliterated. The macula escaped because it was supplied by a cilio-retinal artery, which showed a change in its lumen, but was not blocked. On December 23, 1927, the circulation was completely restored, the white lines along the artery were gone. Even the artery in which the blood-column was interrupted was now restored. Blindness was permanent. The state of the retina three and a half months afterwards is shown in illustration No. 37. Evidently some blood is passing through, because the arteries are not obliterated and reddish in colour. The whitish patch on the disc is obviously the perivascularis, or, more correctly, a scar of hypertrophied perivascularis; it is larger than a trunk even of a normal artery. The same happened to the sup. temp. artery and the white stripes of the degenerated and enlarged perivascularis are still seen at some distance from the disc. Evidently, the white sheaths that I saw covering some of the affected vessels soon after the arterial circulation ceased were the oedematous perivascularis, which later on hypertrophied at the expense of the collapsed arteries. It is of some interest to note that the venous compression by the arteries is not lessened, although the arteries are thin and collapsed.

The interest of this case lies in the fact that it was a purely localised severe atheroma, affecting only one branch of the artery. In essential hyperpiesis the changes are more widely distributed, affecting many vessels—as in the above patient's fundi on January 24, 1926. The important fact is that he had strikingly small changes at the crossings, but deep changes in the lumen of the vessels. One would say that his perivascularis was little affected, but his arterial walls very much indeed. Of interest are the rapid development of thrombosis in a few hours, without any cardiac or cerebral symptoms; the oedema of the retina behind the vessels and along them, but not covering them, as it does in cases of retinitis, especially of renal origin; and the festoon-like line of

division between the areas supplied by different arterial branches.

Conclusions.—Atheroma of the heart or large vessels with normal blood pressure, even in a severe form, does not produce any special changes in the retinal vessels; on the other hand, a severe atheroma of the retinal vessels and even of the cerebral ones may spare the heart and the aorta. Essential hyperpiesis, when added to atheroma, produces marked and advanced retinal changes which depend chiefly on the degree of arterial hypertension.

Involutionary Arterio-Sclerosis and Essential Hyperpiesis

"Old age alone does not produce these changes." *Gunn.*

In the previous chapters I have pointed out so often the very insignificant part played by involutionary arterio-sclerosis of Allbutt, that here I need only summarise. If involutionary arterio-sclerosis were to attack the retinal vessels, retinal sclerosis would be seen much more frequently. After a certain age, involutionary arterio-sclerosis is present in everybody, and yet, although rarely, the retinal vessels may escape completely. If they are frequently mildly sclerotic this is because they can be changed by all toxaemias, whose number is legion, which attack every living being in this world of misery. But if one sees an elderly person with a fundus where the translucency only is lost, with corresponding changes in the light reflex and colour of the vessels, without white lines at the crossings and without arterio-venous compression, one can be sure that the blood pressure is normal. If, on the other hand, with a normal blood pressure at any age, one sees marked retinal sclerosis, it may be either a case of essential hyperpiesis in the past and the blood pressure has now become normal leaving behind permanent changes in the retinal vessels, or some kind of toxaemia was present in the past. Generally speaking, as is clearly seen from group No. 1 of my cases, involutionary arterio-sclerosis may cause loss of translucency of the retinal vessels, white lines at the crossings, and arterio-venous compression. But very rarely is it the cause of silver-wire arteries, change in the lumen of the vessels, or retinal haemorrhages. Essential hyperpiesis will aggravate all these, the silver-wire arteries will appear, the vessels will be changed in their lumen, arterio-venous compression will increase in the number of vessels affected and in intensity, giving often the picture of an "hour-glass" contracted vein, retinal haemorrhages will be present. I have excluded from my cases all those where the kidney efficiency was impaired, as shown by urinary, retinal and laboratory examinations. Atheroma has been described and discussed above. Therefore, we are only left with the unknown toxins of essential hyperpiesis, which attack the retinal vessels (and there-

fore, the cerebral, too) as a spot of predilection as syphilis does in the case of the heart and aorta. In my opinion, *the hyperpietic has a haemorrhage in his head so much more frequently than in other parts of the body, not because the cerebral vessels are mechanically ruptured by hypertension—why then should other vessels escape?—but because those vessels are the ones that are chiefly attacked and are the first to suffer.* It is a well-known fact that in essential hyperpiesis the vessels may remain histologically normal for many years. *As far as the retinal vessels are concerned this does not correspond to the results of my investigation; they lose their translucency early and completely, they change their colour, and " dot " the light reflex—all true histological changes, although so fine that they can be detected only in vivo,* and most probably would appear normal in a microscopical preparation. The connection between the retinal, cerebral and kidney vessels is a well-known fact, but how often do we see haematuria in hyperpietics? I take haematuria as an example because usually a patient is frightened when he passes blood in his urine and is not apt to overlook it and not consult a doctor. I presume here that retinal sclerosis usually means cerebral sclerosis also, but not *vice versa.* This was shown by Hertel and his opinion has not been challenged by anybody until now. Hertel divided his cases into two groups, one of which I call involutionary sclerosis, the purely senile type, and the other, pathological sclerosis. The microscopic changes in the first group were permanent and present throughout the vessel (*in der ganzen Ausdehnung der Gefasse.*) Opposed to that in the second group, the changes were typified by partial localisation (*fleckweise auftreten.*) They were met with only at localised points and *chiefly in the cerebral vessels.* In the retinal vessels they were very small, and very seldom were they seen with the ophthalmoscope. Am I justified in saying that my clinical conclusions coincide with those classical post-mortem findings of Hertel? Ramsay, p. 397, says " *arterio-sclerosis* may show itself very early in the retinal blood vessels "—I should say—*essential hyperpiesis.* The same applies to the sentence of Foster Moore (p.50). " In general arterio-sclerosis the retinal arteries, of course, share . . . consequently arterio-sclerosis is one of the diseases often first discovered by the ophthalmologist." It is the chief purpose of this paper to affirm that in general involutionary arterio-sclerosis the retinal vessels are affected only very moderately, or not at all, and Foster Moore is correct only if he means essential hyperpiesis. The same applies to Parsons' opinion (*Path. of the Eye,* p. 1271) " General arterio-sclerosis manifests itself by changes in the retinal vessels—often the first observable sign." And farther on (p. 1281) " The cerebral vessels are sometimes highly sclerosed, although no

evidence of general arterio-sclerosis is furnished by the peripheral arteries." This coincides entirely with my view if only essential hyperpiesis is meant instead of "general arterio-sclerosis."

When Allbutt first characterised essential hyperpiesis as accompanied by the "Sergeant Death," the medical profession was most probably staggered. Since then the physician has found that the fatal end is not always near at hand, even with advanced essential hyperpiesis and the pendulum, it seems to me, is swinging to the other extremity—to regard arterial hypertension more or less light heartedly. This is very wrong indeed, since it would be comprehensible for the lay-mind, which by the term "danger" understands something serious for to-day or to-morrow. The insurance companies are the best judges and their publications leave no doubt on this matter. "Of 525 applicants accepted in earlier years who showed an average systolic pressure of 152.88, the N.W. Life Insurance Society suffered an excess mortality exceeding by over 30 per cent. the general average of that company. In another group of 1,970 lives, showing an average systolic pressure of 161.44 and representing the rejected risks, followed only in part and under great difficulties, the mortality rate was almost two and a half times greater than the general average of the company. Yet, of this group, at the time of examination, 1,082 showed no other impairment, than this relatively high systolic pressure and the individuals composing this subgroup and showing but the single blemish (B.P. 161.44) yielded an excess mortality more than double that properly to be expected." (Green, p. 478.) A medical man must always be on his guard and thoroughly examine a hyperpietic. And I earnestly affirm not only to the deus minor—the general practitioner, but the *dei majores olympici* of the profession that the ophthalmoscope, when properly used, will give very precious data, more cheaply and more quickly and even more certain sometimes than very complicated, costly and delicate auxiliary examinations, often outside the scope of an all-round consultant. And I gather from Fisher's publication that no ophthalmoscopic examinations were made by the "dei" of the insurance company.

If this fundamental idea were grasped, one would never cease to use the luminous ophthalmoscope in routine examinations. From time to time, I come across cases, seen by hospital and private medical men, crying out, so to speak, for ophthalmoscopic examination, which immediately reveals the seriousness of the situation. There is always hope for the man who acts, even though he makes *bona fide* mistakes, he will learn by them; but there is no hope at all for the one who does not act because he is afraid of making a mistake. Important is the revision, the reaction in the medical mind towards the ophthalmoscope; the

inertia and piety of to-day must go, the instrument must be used more often and its results appreciated more widely.

In *Brit. Med. Jl.* of March 3, 1928, appeared an article by R. Hutchison on the principles of diagnosis. His views are very much the same as mine. Clinical observation is eclipsed nowadays by laboratory examination. The leader, on p. 38, does not agree with this. " But laboratory methods do provide facts; and it is here that the clinician is sometimes at fault, for if the laboratory finding is not in harmony with his clinical conception of the case he is apt, especially in his teachings, to draw distinctions between " clinical observation " and " laboratory methods," which are by no means flattering to the latter. This is all wrong; there is no real distinction between the two types of observation. . . . The fact that a patient has . . . a raised blood urea or a positive Wassermann reaction is merely a clinical observation. . . ." It all sounds very clever in theory, but let me tell of a fact. A young woman aged 31 years, married a man whose first wife had died from phthisis a few weeks after her confinement, leaving a healthy baby. In May, 1927, when six months pregnant, she coughed up a mouthful of blood. Alarmed, she consulted her local doctor who wisely referred her to a well-known special hospital. Her temperature was constantly normal, her sputum negative. But an X-ray of the chest showed an active tuberculous process and she was advised to have her pregnancy immediately interrupted. She protested vigorously and was allowed to wait another week or so. At the end of this time, though the temperature was normal and the sputum negative, another radiogram showed that the process was spreading. She was then transferred to a large teaching hospital, examined afresh and again strongly advised to get rid of the foetus. Still dissenting, on the eve of her undergoing the operation, she consulted another local doctor who could not detect any physical signs in her lungs. The next day she saw a careful and elderly consultant, who agreed with the practitioner that she could be allowed to go to full term. She gave birth to a healthy baby—an anaesthetic being necessary—and is now, a year after her confinement, in good health, the child being likewise. Throughout this time she has not shown any clinical signs of pulmonary tuberculosis; normal temperature, no cough, putting on weight. The reader is asked to believe me, that I am very well aware of the dangerous character of haemoptysis in a pregnant woman!

Diabetes

In April, 1927, a man over 50 years of age was suddenly seized with vomiting, severe pain in the stomach, and signs of intestinal obstruction. After a day or two of agony, on the advice of two

local practitioners, he proceeded to a large teaching hospital for a laparotomy. After being admitted to the hospital he was examined by a surgeon, who also made a diagnosis of acute intestinal obstruction and advised an operation. Next morning, the third day of his illness, the patient was brought to the anaesthetic room and refused to allow an operation. The angry surgeon discharged him. At midnight of the same day he was seen by another practitioner: Temperature 96°F.; pulse 121; blood pressure 120/70. Soft, painless abdomen; meteorism. Black, furred, badly smelling tongue. Agonising pain in the upper part of the abdomen. Facies nearly Hippocratica. Extremely soft eye-balls (by palpation). Glycosuria present, acetone bodies absent by the ferric chloride test. Insulin in large doses injected. In five days the man recovered completely. In a month, insulin was stopped. Now, eighteen months after this attack, he is allowed to eat starchy foods freely, but no sugar or honey. He is sugar free, he has put on weight, his blood pressure is 160/90, his fundi are mildly arterio-sclerotic. How to label his case I really do not know. Very vaguely it may be described as an acute disturbance of the endocrine activity of the pancreas, but this does not help us. The absence of acetone in the urine does not alter the diagnosis of diabetic coma. Weichmann points out this fact, and, in his opinion, the findings in the urine are not decisive. Very nearly of the same opinion is Docent Elias, who shows that the amount of sugar and acetone in the urine may be very small, if the patient eats nothing and the vascular system is impaired.

In spite of tremendous progress since Banting's discovery, there are still many obscure points in our theory of diabetes. Is the blood pressure raised by the diabetes itself or by some secondary products of the faulty metabolism? Why are some cases refractory to insulin? What is the influence of insulin on the ocular changes? These and many other important questions are still undecided.

I wonder if a student would escape without grievous harm from his examiner if he were to omit the description and discussion of eye changes in his account of diabetes mellitus. And I am sure that many of the examiners were present at last year's conference of the British Medical Association, when McLean opened a discussion on diabetes mellitus and insulin. He divided diabetes into the true and the false variety; the former is a true disturbance of pancreatic activity and is accompanied by ketonuria—the second one, chiefly a disease of the aged, is simply an inability of the tissues of the body, especially the muscles, to utilise all the glycogen which is liberated by the liver. A lively discussion followed and only one speaker as an aside mentioned the importance of the ocular changes.

In *Brit. Med. Jl.* of May 7, 1927, is published a very good paper by Earl, of the Salford Royal Hospital, on the treatment of diabetics with insulin in the out-patient department. The utmost care is taken, the weight is recorded, the blood often tested for sugar, but the ophthalmoscopic examination is not mentioned at all. I presume it is not done. Minkofsky, in his paper, as far as I am aware, also does not pay any attention to the importance of the ophthalmoscopic examination.

And it is so important and helpful. My analytical table shows the average age as 50, males 12 cases, females 14, average blood pressure 188/98; retinal sclerosis marked and advanced. I have never seen a diabetic with the translucency of his retinal vessels preserved. How helpful the ophthalmoscopic examination may be is shown by the following two cases.

A man aged 52 years, suffered from diabetes two years ago. Presented himself now complaining of general malaise. Urine normal, sp. gr. 1015, blood pressure 140/70. Fundi: translucency lost, copper-wire colour, light reflex dotted. White lines on the disc and crossings. The right sup. nasal vein going upwards from the disc passes over a focus of old chorio-retinitis; the vein is very narrowed at this point and much banked above it. Many spots of old chorio-retinitis, corresponding to the lower temporal branch of the lower artery. In the central part of the retina, especially near the macula, many fresh spots of chorio-retinitis. Nothing special in the left eye. Following these findings, his sugar tolerance (not only the blood sugar) was tested. His fasting sugar was 0.105. Then 50 grammes of glucose in water were given to the patient; after 45 minutes his blood sugar rose to 0,180, glycosuria present. After two hours, glycosuria absent, blood sugar 0.095. Diagnosis: mild diabetes mellitus. Dieting improved his general health and also the eye condition.

Transitory myopia and symmetrical ulcers in the cornea have been described by Rosenstein, in the early stages of diabetes.

I do not like the term "retinitis diabetica." Retinitis implies, first of all, a hyperaemia, *an oedema of the retina*. And, as far as my experience goes, I have never seen this in pure diabetes. The exudation in the retina is clearly defined, not of such a "fat" colour as in nephritis; often surrounded by pigmentation; the vessels are clearly seen, although they may be markedly degenerated and haemorrhages may be present. With some training one can differentiate these changes from renal ones, although this is sometimes extremely difficult. Still, the presence or absence of retinal oedema would, in my opinion, be a deciding factor; the oedema is characteristic of nephritis.

A woman, aged over 50 years, was seen at the ophthalmic out-patient department of the London Jewish Hospital, on April 15,

1927. The diagnosis of diabetes mellitus was made two years previously in the medical department, and she was treated accordingly. Six months before she was admitted to a large teaching hospital for a minor operation and was told that she did not suffer from diabetes. Her eyes were not examined there. Fundi: translucency lost, copper-wire colour, light reflex dotted. The veins are crushed by the arteries and deflected centrifugally. Many small haemorrhages. Many old and fresh diabetic exudations, especially in the right eye, where a star is formed. Blood pressure 225/125. Urine: sp. gr. 1020, sugar ++, albumen +. The reader will no doubt agree that if the fundi had been examined at the large teaching hospital the diabetes would have been detected, in spite of the negative urine findings.

On April 26, 1927, her blood sugar was 0.287 and her blood urea 49 mgm. On June 14, 1927, her blood sugar was 0.26, and her blood urea 27. Urea concentration test:

Before urea	1.8 per cent.
One hour after	2.1 per cent.
Two hours after	2.1 per cent.

She was an obvious case for immediate insulin treatment. But she disappeared. She was seen again on February 10, 1928. Fundi: the same, " star " around the macula in the right eye. Small round haemorrhages in both eyes, discs clearly defined. No oedema of the retina present. Urine: sp. gr. 1010, albumen +, sugar −. Still I adhered to my diagnosis of diabetes because retinal oedema was absent, although the blood pressure was the same. The following week came the result of her blood test: Blood sugar 0.259, blood urea 42 mgm.

Her urea concentration test was:

9.30 a.m. (before urea) ...	1.9 per cent.
(urea given immediately afterwards).	
10.15 a.m.	1.4 per cent.
10.40 a.m.	1.3 per cent.
11.15 a.m.	2.0 per cent.
11.45 a.m.	2.1 per cent.

In my opinion, she was a case of diabetes with benign nephrosclerosis.

The following case is more complicated. She was described in my first paper under No. 6 C. Her blood pressure rose from 150-160/90 to 220/100, in spite of insulin injection. She

developed severe sclerosis of the retinal vessels, with many extensive retinal haemorrhages and foci of exudation all over the retina. Fine oedema of the retina. The specific gravity of the urine was 1005. The vision was very much impaired, as the retina was atrophied in many parts. This was in the autumn of 1926. Since then her blood pressure was 185-210/80-100. She still had extensive haemorrhages in her retina, but otherwise she was quite well, active, walking about, and doing her housework. On February 12, 1927, I saw in her right eye a branch of the lower ret. artery accompanied by a large area of white exudation, so that the artery was partly covered by it, narrowed and deflected. Other vessels were crossing over their exudation spots, unchanged and clearly seen. On May 7, 1927, the picture was the same, but in some places the vessels were covered by the exudation. On November 3, 1927, her blood pressure was 230/100. Fundi: unchanged, but there was a striking change in the lumen of the vessels, especially of the veins. Her blood urea was 49 mgm. per 100 c.c. blood. Phenolphthalein test 6.6—22.2—4.3—; urea concentration test 1.1 per cent. and 1.7 per cent. On November 23, 1927, with a blood pressure of 225/100, she complained of retrosternal pain. Urine normal, sp. gr. 1000. Fundi: exudation and oedema of the retina disappeared. Haemorrhages, old and fresh. Atrophy of the retina. The physician, under whose care she was in the hospital, kindly wrote to me on November 4, 1927. " I first saw her on January 7, 1924. There was glycosuria with a blood sugar of 0.36 per cent. She was admitted and remained sugar-free on a diet of 1,700 calories, being discharged after five weeks with a blood-sugar of 0.133 per cent. In May, 1927, she was again seen as an out-patient with glycosuria and a fasting blood sugar of 0.077 per cent. The first note of her blood pressure is in May, 1925, when it was 240/120. There were then hard radial arteries and the fundus showed silver-wire arteries, exudate and many old haemorrhages. In May, 1926, she was sugar-free with a blood-sugar of 0.22 per cent. and I put her on insulin, 5 units twice a day. In August, 1926, the blood-sugar was 0.1 per cent. In October, 1926, the retina showed recent flame and dot shaped haemorrhages and a spot of chorio-retinal atrophy."

A few interesting conclusions may be drawn from this case. We can exclude diabetes as the cause of her hypertension and retinal changes. She is on insulin, her blood sugar is normal, the urine is free from sugar, with a specific gravity of 1,000-1,005. Then we are left only with essential hyperpiesis and interstitial nephritis. The retinal changes are extremely severe; the most important point is, in my opinion, the oedema of the retina and the exudation not only behind, but also over the vessels. Unfortunately, when the retinal condition was at its worst, the hospital

did not test the efficiency of her kidneys, and a good opportunity was lost. The diagnosis lies, therefore, between benign and malignant nephrosclerosis. Considering carefully her retinal changes, I am inclined to think that she started as a case of interstitial nephritis (malignant nephrosclerosis) and the process now is either proceeding very slowly or is arrested altogether. That the last suggestion may prove to be the correct one is supported by the statement of Leber, p. 817, that " the retinal changes in true or albuminuric retinitis can recede if the basic pathological process in the kidney is healed or improved. In good cases even highly advanced retinal changes can recede completely." Here lies the chief interest of this case. Again, in considering this case, a conclusion presents itself that there is some intimate connection between the retina and the efficiency of the kidney (endocrine?). More than half of her vision is gone, more than half of her retinae is atrophied. If she would bleed to the same extent in the brain (I do not even say the lungs, etc., where the haemorrhage would be visible, so to speak, to the naked eye) she would surely be half paralysed or her mental capacity would be affected. And it is not. Even her hearing is not affected, except perhaps for slight giddiness, which is common to every hyperpietic of her age and condition. Why then, of the whole of her brain, have only the retinae suffered so terribly?

The third case was fully described as No. 4 C in my first paper. Briefly, the particulars were as follow: Kept on insulin for three years. Her medical adviser thought that she suffered from " cataracts " because her vision was going. When seen by me, she presented haemorrhages in the retina, atrophy and fine oedema of the retina. Arterial hypertension. Two months later her blood pressure was 164/70. She died a few days afterwards, most probably from uraemia.

The connection between diabetes mellitus and nephritis has long been known to oculists. Leber writes, on p. 898, that retinitis, after the healing up (*Ruckbildung*) of diabetes, is sometimes caused by nephritis. He goes even further than that, and on p. 915 he makes a very important statement, that with a large degree of probability we can assume that the disease of the retina starts only when the disturbance of the function of the kidney has caused already a rise of blood pressure. Yes, for the retina; no, for the retinal vessels, I should say. We shall see the difference immediately, when discussing the problem of nephrosclerosis.

On p. 949, he states that nephritis especially must be reckoned as the cause of retinitis, when the latter is of characteristic appearance and especially when the glycosuria is diminished or absent altogether.

Conclusions.—Even mild diabetes attacks the retinal vessels, but most probably to the same degree as the vessels are affected in the other parts of the body. Constant, frequently repeated, and careful supervision of the urine, blood pressure and retinae is necessary in every case of diabetes, be the treatment purely dietetic or by insulin; aggravation of the retinal changes, haemorrhages and especially oedema are often combined with rise of blood pressure and lowering of the specific gravity of the urine.

Nephrosclerosis

Many fascinating problems of importance immediately suggest themselves from the name of this chapter. But I am obliged in this paper to discuss only sclerotic retinal vessels, and shall therefore keep within those limits. The problem of the co-ordination or correlation between the kidney and the retina is too complicated and important to be treated only in a few lines. In the meantime, I shall discuss this question from the point of view of sclerotic vessels.

There are two items in my analytical tables, which I have purposely not discussed: Blurred disc and exudation on the retina. Again, as in the case of diabetes, I do not like the term of "sclerotic retinitis." Those exudations are usually not accompanied by oedema of the retina. They are usually small, sharply defined, and often, but not always, in the immediate neighbourhood of a vessel. They never cover the vessels, as usually occurs in renal retinitis. The vessels are clearly seen, they are sharply demarcated from the surrounding retina. Foster Moore also describes them in his "Medical Ophthalmology." They are already on the verge of retinal degeneration and are therefore not included in the theme of this paper. It is important only to note that they may disappear, sometimes in a few weeks, without leaving any traces, whilst the vascular changes, as has been pointed out many times previously, are always permanent. The parenchyma may improve, the vessel does not. This is in accordance with one of the most noteworthy laws of pathology, which applies to the retina as well as to any other organ of the body, that *there is no parallelism between vascular sclerosis and changes in the parenchyma.* This is also the opinion of Leber who quotes Opin and Rochon-Duvigneaud (p. 870). In spite of advanced endarteritis, an organ can function pretty well. And the visual acuity of the retina is usually not impaired even by advanced sclerotic changes. Even an advanced sclerotic kidney, primary nephrosclerosis, benign nephrosclerosis, arterio-sclerotic

kidney, call it what you please, will allow a good deal of excretory activity and will not endanger the well-being of the whole body to any excessive degree. On the other hand, in a severe parenchymatous degeneration, when the function of the organ is considerably and dangerously impaired, the vascular changes may be insignificant. In albuminuric retinitis the vessels may be nearly normal (Ginsberg). True interstitial nephritis—I should not like to be misunderstood, I do not possess more knowledge of its nature than anybody else, and altogether we know very little—is accompanied by rapid and tragic lesions of the whole body. Involutionary arterio-sclerosis implies only limitation of function; essential hyperpiesis is much more dangerous; interstitial nephritis (malignant or secondary nephrosclerosis) means a quick and tragic end. In the retina the first *per se* is seldom seen, and may be absent altogether; the second one means sclerosis of the perivascularis, change in the lumen of the vessels, haemorrhages and exudation; the third is accompanied by inflammation of the parenchyma, oedema, and its special form of exudation. For dialectic purposes I have described all of them separately; in life they are often combined, supplementing and replacing one another, making a clear distinction difficult and sometimes nearly impossible. However, this outline is very helpful for prognosis in certain cases. *Caeteris paribus*, the fate of the hyperpietic depends upon the degree to which his toxaemia is developed, his kidney is involved and its efficiency impaired. *One cannot, however, rely upon the blood pressure, the retinal picture, or the kidney efficiency tests alone*, in the prognosis; they must all be combined. A man with blood pressure 210/140 may be much nearer his end than another man with blood pressure 250/130. Retinal haemorrhages are less important if the blood pressure is steady, but if they increase, in spite of the blood pressure being steady or even going down, the outlook is gloomy. In my experience, the silvery-wire and tortuous independent small vessels around the disc are a bad omen. But worst of all is oedema of the retina. I do not mean by this retinitis albuminurica. The former is simply a fine veil, covering the vessels on the retina. One day you examine a fundus, well-known to you, and somehow you cannot see clearly the vessels. Changing the refraction does not help, nor increasing the beam-light. The vessels are covered and imbedded in a liquid. Sometimes it takes a few days, sometimes a few weeks, and this treacherous veil disappears to come again and again, playing with its victim, like a cat with a mouse. Sometimes the patient himself will tell you that he sees "foggy." This oedema is a sure sign that the kidney efficiency is impaired and that the end is approaching. "Some degree of oedema of the retina is probably present in all cases," says Foster

Moore; I entirely agree with this and emphasise it, but one has to examine the patient frequently and carefully. In the great majority of cases the kidney efficiency test, like the Wassermann reaction for syphilis, will coincide with this oedema and other clinical findings; in the minority it will differ. Theoretically, of course, the leader in the *British Medical Journal* is correct. The kidney efficiency test is also a clinical feature, but with our present imperfect knowledge the practical result will be different. "This patient is not suffering from malignant nephrosclerosis, his kidney efficiency tests being good; he suffers only from a gross cardio-vascular lesion, and therefore the prognosis is not so bad," will say the laboratory worker. "You are wrong, Sir," modestly whispers the retina, "You are testing the kidney only for the toxins that you know, but there are others that you do not know, and they are poisoning me." Two cases will illustrate this.

A woman aged 65 years, an excessive eater, developed in 1923 acute intestinal obstruction. A laparotomy was performed and, in addition to the obstruction, the surgeon discovered a gall-bladder full of stones. It was not possible to remove it immediately; the woman recovered from her intestinal obstruction. She was discharged from the hospital and admitted again a few months later for an operation for gall-stones. She bore it quite well. In 1926 she was admitted into the medical wards of the London Jewish Hospital, suffering from pyelitis and cystitis, her blood urea being 250 mgm. Her systolic blood pressure was 195. As medicinal treatment failed to relieve her, the surgeon, Mr. Sourasky, decided to make a permanent suprapubic fistula. I saw her a few days after this operation with a blood urea of 175 mgm. Fundi: translucency absent, light reflex dotted, copper-wire colour, white lines at the crossings. The veins were crushed by the arteries with interference to the blood-stream. Some arteries showed a change in their calibre. No haemorrhages, no oedema. Blood pressure 105/65. Five weeks afterwards she was discharged from the hospital with blood urea 84 mgm. Fundi were the same, having been examined each week. A year afterwards I saw her again, she was now aged 70 years. Her blood pressure was 140-150/75-80, her fundi were the same, the veins being more crushed, her blood urea was 62 mgm. per 100 c.c. blood.

The second case was No. 17 C. of my first paper. I saw in her, on a few occasions, fine oedema of the retina. Her blood pressure was 250-300/130-160, in spite of medication and dieting. One kidney efficiency test gave "blood urea 17 mgm. per cent.; urea concentration test 0.4 per cent.—0.7 per cent.—0.8 per cent." The test was repeated many times by a prominent research worker in this country. He declared that her kidneys were fairly good.

Under pressure of the relatives, whom I warned of the gravity of the situation, I communicated with him. He kindly answered me: "She is really suffering from gross cardio-vascular changes. Her kidneys are by no means badly affected and any change that is present in them must be attributed to the primary arterio-sclerotic condition. I think that the eye changes seen in the majority of renal cases are due to the cardio-vascular changes which usually accompany the disease and not to the renal lesion. I have often seen a case where chronic nephritis was diagnosed by examination of the eyes and yet the patient's kidneys were apparently quite healthy, though he always suffered from cardio-vascular changes. I am quite certain that it is impossible to diagnose between high blood pressure alone and renal disease by eye examination. Mrs. X's kidneys are comparatively good. Her blood urea is normal and her urea concentration test quite satisfactory. If her kidneys were bad she would be dead long ago." And so she was ten months after this letter to me was written. She went out for a walk, being in "good condition," returned home and immediately died, sitting in a chair, most probably from a cerebral haemorrhage. I was away from London at the time, and I did not see her during the last few months. But the above was the impression of the doctor who saw her dying. Nevertheless, a gifted man is admirable even in his mistakes; as in a nutshell, in the letter quoted above, is put the whole controversy. If the retinal changes are only secondary to the vascular changes he is correct; if they are primary—an actual disturbance of the retinal metabolism as a result of the kidney intoxication—he is wrong. I have dealt at length with this difficult question in my first paper. Here I shall briefly state that the retinal changes are primary, and that my correspondent is wrong. That is why I have discussed throughout the present paper only the sclerotic vascular changes and have abstained from discussing sclerotic changes of the retina itself, and more so retinitis—they are different clinical entities, with different aetiologies and different prognoses, although they may co-exist.

It is a remarkable fact that this oedema of the retinae in my experience, is always connected with arterial hypertension, but there is no oedema of the subcutaneous tissue unless the heart is commencing to fail. On the other hand, I have seen in my life not a few cases of serum-sickness, and the oedema of the subcutaneous tissue, all over the body, but especially of the face was sometimes very considerable indeed. There was usually also a good deal of albumen in the urine, but I never noted a rise of blood pressure, nor oedema of the retinae, nor even a blurred disc.

And the kidney is still a very little-known and treacherous organ. Only recently I deplorably failed to recognise

an early stage of uraemia. I was called to see a woman aged over 50 years, thin and miserable looking, with chronic bronchitis. She was afraid to take food, quarrelled with the family, was sleepless and excited at nights. Her blood pressure was 160/90, traces of albumen in the urine, sp. gr. 1010. Fundi, as far as I could examine at the patient's house, in a darkened room, and with undilated pupils, were mildly arterio-sclerotic. She complained of severe epigastric discomfort, but no headache, giddiness, or disturbance of sight. A diagnosis of some mental or hysterical excitement was made. As she did not improve, I sent her to a special hospital where the diagnosis of delusions was confirmed and admission to the Infirmary was suggested. She died three days afterwards in a state of uraemia. Post-mortem showed a very small contracted kidney with a very poor cortex and a partial thrombosis of the basal cerebral artery.

The blurred disc, when seen in hyperpietic cases, does not imply, in my opinion, anything special and dangerous. I speak, of course, of the mild form only. I simply think that the condition of the circulation is very unfavourable in the sclerotic vessels, compressed as they are on all sides, and, therefore stasis of the lymph is more easily produced. Larson points out the fact that even a " choked " disc may occur in cases with high blood pressure, without there being clinically any demonstrable renal disease.

What is the nature of hyperpiesis? Is it a disease *per se*, as described by Allbutt, or is it simply a protracted initial period of Bright's disease, interstitial nephritis? The dispute does not seem to be settled even now. Lyon, p. 766, is the advocate of the second theory; " In many cases, the syndrome of arterial hypertension well described by Dieulafoy under the name of minor signs of " Brightism," may develop with little of the symptoms of proper Bright's disease. . . . This cardio-vascular form constitutes also the first stage, often a long one, of Bright's disease, before it ends in the syndrome described under the name of interstitial nephritis." In German speaking countries, Pal is of the same opinion as Allbutt, and, if I am not mistaken, Volhardt is also. However, Kylon testifies that in the hospitals and clinics of Scandinavia and Germany the view still prevails that essential hyperpiesis is really an early nephritis. He himself thinks that essential hyperpiesis is not a kidney trouble at all, but a disease of the vegetative nervous system with the influence of the vagus being predominant over that of the sympathetic. In this country Batty Shaw is of Allbutt's school; the best résumé was made at the meeting of the Manchester Pathological Society. Maitland Ramsay, p. 397, writes " Arterio-sclerosis may show itself very early in the retinal blood vessels and give rise to haem-

orrhage long before there is any manifest renal inadequacy. It is prudent, however, to regard those cases as pre-albuminuric."

I have dealt at length in this paper with 269 cases whose average blood pressure was 200/110. It means that in a large proportion of them, roughly a third, the systolic blood pressure was 230-250 and the diastolic 110-140. In not one of them, although a few have been known to me for at least five years, was there any clinical evidence of renal impairment, as shown by the blood-urea test, frequent and careful retinal examination, testing of the urine and minute clinical investigation. Therefore, there is no reason whatsoever for considering that they represent the early stage of Bright's disease. Moreover, the retinal condition does not suggest it either, and I have purposely excluded all cases with oedema of the retina, even in its slightest degree. I agree with Lewandofsky when he considers that every systolic blood pressure of 180-200 or higher means a diffuse vascular nephritis, most often an arterio-sclerotic kidney. But I should reverse the sentence of Green: "*As a matter of practical application a decided and persistent hypertension with a high or relatively high diastolic level means chronic nephritis, until another cause is proved or rendered a reasonable assumption.*" (Italics of Green.) In my opinion, it means first of all essential hyperpiesis, until chronic interstitial nephritis is proved or rendered a reasonable assumption.

Extravaganza

"*Dans la nuit, ou nous sommes tous, le savant se cogne aux murs, tandis que l'ignorant reste tranquillement au milieu de la chambre*" (Anatole France). It is quite possible that when I was basking in the green light of my luminous ophthalmoscope and thought myself firmly established on the membrana limitans interna retinae, examining the details of vascular sclerosis, I was really suspended somewhere in the centre of the corpus vitreum at the end of one of its fragile fibres. But at least in the preceding chapters I have been able to show facts that could verify every word of my reasoning. Here, in the concluding chapter, I repudiate such a responsibility and shall make some general remarks rather of a fanciful nature, for which evidence should not be asked.

We have seen that sclerosis of the retinal vessels is produced chiefly by essential hyperpiesis; that it is represented by sclerosis of the perivascularis and pathological changes in the vascular wall itself. This sclerosis of the perivascularis tissue together with the change in the vessel wall is highly characteristic of essential

hyperpiesis. It does not correspond entirely to fibrosis, but it is something very similar. It is probably a defensive reaction, an attempt of the vessels to carry on their vital function as well as they can under pathological conditions. And this is in accordance with the whole picture of arterial hypertension, which is, after all, a defensive reaction of the body, a reaction which is usually overdone and is damaging in its effects, somewhat like the diarrhoea of infants. It means that the interstitial tissue degenerates and, contracting, squeezes in the vessels, compresses them. It would only be logical to look for an analogy in other parts of the body, the anatomy of which is better known than that of the retina. In the liver, and especially in the kidney, we shall find a very similar process. " In cases of albuminuric retinitis, post-mortem shows a contracted kidney, diminution of the organ, considerable overgrowth (*Wucherungen*) of the interstitial tissue and atrophy of the secreting parenchyma, especially of the cortex." (Graefe-Saemisch, p. 895.) In Price's text-book we find the following definition of chronic interstitial nephritis: " A fibrosis of the kidneys with degeneration of glomeruli and tubules, associated with raised blood pressure, hypertrophy of the heart and abundant urine." And lower down: " The vessels of the brain are particularly apt to be diseased, hence the special liability of such patients to die from a cerebral haemorrhage." Anders does not express quite the same opinion: " A chronic diffuse inflammation of the kidneys, attended with a growth of connective tissue in the stroma, degeneration and atrophy of the parenchyma and marked cardio-vascular changes. The destruction of the renal parenchyma is due to the circulation of noxious agents, and it is replaced by cicatricial fibrous tissue (Weigert)." He wisely differentiates four forms; the primary one, the arterio-sclerotic kidney, the senile type, and the secondary form. I think this is a very good definition and meets the case better. If the parenchyma is primarily attacked and destroyed, we have the secondary kidney, malignant nephro-sclerosis—and that is why the clinical picture is of an acute and severe poisoning, with very considerable danger to life; but if the parenchyma is only squeezed, compressed by the degeneration of the interstitial tissue, secondarily involved, we have primary sclerosis of the kidney, benign nephrosclerosis. We shall get here the picture of essential hyperpiesis. Ultimately, of course, both forms will meet *in extremis* because whether primarily or secondarily affected, the parenchyma will go. Thus we see in the case of kidney how correct is the rule mentioned previously that an organ can function satisfactorily, although its vessels may be in a state of advanced sclerosis. The clinical picture is the same for patients with highly advanced retinal sclerosis and primary kidney (benign nephrosclerosis)

raised arterial pressure, especially in the diastolic one; low specific gravity of the urine; age over 40-50 years; restlessness and irritability of the nervous system—all pointing to some toxin, circulating in the blood and attacking the perivascular tissue and the vascular wall, but relatively sparing the parenchyma.

We have seen that for the retinal vessels essential hyperpiesis means sclerosis of the perivascularis. The retinal perivascularis contains the lymphatic vessels. If the perivascularis of other vessels also contains the lymphatic vessels, then this form of essential hyperpiesis is connected with toxins chiefly circulating in the lymph. The lymph is produced in the intestinal tract and the cells of the body, hence these toxins, in great part at least, are produced in the intestines either as by-products of metabolism or as the result of the bacterial flora. We already know that guanidine and cholesterine produce a rise of blood pressure in the vessels. Metchnikoff also pointed out the importance of chronic intestinal auto-infection in the premature ageing of mankind. And there is no doubt that arterial hypertension means the shortening of life. On the other hand, there is another form of vascular degeneration where the wall itself is the chief sufferer, fortunately in the minority of cases. And about the toxins of this form very little is known at present. The retina stands firm and fights long and hard against this intestinal toxaemia—essential hyperpiesis; it succumbs quickly and shows the affection very early when it is attacked by other toxins. Some of them, although circulating in the blood, produce benign nephrosclerosis. The others, perhaps produced chiefly by faulty metabolism of the body's own cells, but certainly associated primarily with bacterial infection, are intimately connected with impairment of the kidney and produce in the retina the characteristic retinitis albuminurica. Different toxins attack various parts of the vascular tree. To discover them, to trace them, is an enormous difficulty, but therein lies the thrill, the interest, and the reward of medical research. Unfortunately, the reward is seldom obtained because we are only beginning this new line of scientific investigation, and " life is short, art is difficult and the way uncertain."

It is my pleasant duty to express my sincere gratitude for the permission to use their drawings for the purpose of this paper to Prof. Krückmann (No. 1-1c), Mr. Ernest Clarke (No. 2), Mr. Dent (No. 3), Mr. R. Batten (Nos. 4, 7, 11, 14, 16, 18). Mr. Beaumont (No. 5), Mr. Thomson (No. 6), Mr. Joseph (No. 8), Mr. Summers (No. 9), Mr. A. H. Levy (Nos. 10, 12, 26), Mr Ormond (No. 13), Mr. Marshall (No. 15), Mr. Goulden (No. 17), Mr. Davenport (No. 19), Mr. Evans (No. 25) and Mr. R. Junk (Nos. 31, 32).

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ANALYTICAL TABLE.

	No. 1 Group. (169 cases). Average age 47 years 4 months. Average blood pressure $\frac{128}{78}$		No. 2 Group. (269 cases). Average age 52 years. Average blood pressure $\frac{200}{110}$.		Diabetic Group. (26 cases). Average age 50 years. Average blood pressure $\frac{188}{98}$.	
	Cases	%	Cases	%	Cases.	%
Translucency diminished	2	1¼	—	—	—	—
Translucency lost	167	98¾	269	100	26	100
Light reflex dotted	128	76	256	95	26	100
Copper-wire colour	133	79	256	95	26	100
Silver-wire arteries	1	½	44	16	2	7
Change in the lumen of the vessels	7	4	65	24	6	23
Tortuosity of the vessels increased	1	½	25	9	2	8
Veins depressed only	13	7	18	7	2	7
Veins depressed and deflected	11	6	14	6	1	4
Veins crushed with interference with the circulation	20	12	108	40	5	20
Veins crushed without interference with the circulation	24	14	48	18	9	35
Veins crushed and deflected centrifugally	29	17	97	36	10	40
Veins crushed and deflected centripetally	15	9	37	14	5	20
White stripes on the disc	78	46	122	45	11	4
White stripes on the crossings	74	44	178	66	17	65
White stripes on the retina only	3	2	11	4	1	4
Narrowing of the vessels	4	3	15	6	1	4
Discs are not sharply outlined	2	1	10	4	—	—
Oedema of the retina	—	—	2	1	1	7
Old retinal haemorrhage	1	½	7	2½	2	4
Fresh retinal haemorrhage	—	—	25	9	4	15
Exudation on the macula	2	1	8	3	1	4
Exudation on the retina	—	—	8	3	2	7
White stripes between the vessels	4	3	13	5	1	5
Aneurysm on the retina	1	½	3	1	—	—
Aneurysm on the disc	1	½	1	½	—	—

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A HAEMANGIOMA OF THE MENINGES INVOLVING THE VISUAL CORTEX

BY

R. FOSTER MOORE

LONDON

JOHN B., aged 12 years, was sent to me on September 12, 1928, by Dr. H. W. Beach, of Hassocks, on account of defective vision of his left eye. The parents' particular concern was as to the likelihood of the right eye becoming affected, and whether he should go to school in the ordinary way. He had seen Dr. E. V. Oulton, of Hove, in 1925, when his visual acuity, with a small hypermetropic and astigmatic correction was 6/9 in the right eye, and 6/12 in the left. It was stated that the left eye had got worse during the last year. The boy's father is said to be blind from locomotor ataxia.

He was a healthy-looking boy, and complained of nothing except defective sight of his left eye. He had a naevus of the left half of the face involving the eyelids. The left pupil reacted very poorly to light; the visual acuity of the right eye with correction was 6/9 partly, and that of the left 6/6 partly. There was a slight congenital dislocation of the lenses, and a small coloboma of the left optic disc. Otherwise there was no discoverable disease in the eyes themselves.

The fields of vision showed that not only the left eye, but the right eye also was affected; he had, in fact, as will be seen by the charts, an incomplete right homonymous hemianopia (Figs. 1 and 2). No previous charts were available, and so it was not possible to say whether the defect had in fact progressed within the last year, as was stated.

With regard to the cause of the lesion; in the absence of evidence of any of the common or likely causes of such a lesion such as cerebral tumour or abscess or vascular disease, and having regard to the naevus of his face and the congenital defects in both eyes, I said, in writing to Dr. Beach the same night, that I thought the likeliest cause was a naevus of the visual cortex, and suggested that a skiagram of the skull should be taken to see whether this would provide us with any fresh evidence. This was done by Dr. Prowse, and, as will be seen, the evidence is most striking. There is a tumour composed of a new growth of large vessels, evidently a haemangioma of the meninges. This tumour, when seen in a lateral skiagram, is acutely wedge-shaped (Plate I), the apex being far forward in the temporal region and apparently being situated quite superficially under the bone in this region, as is seen in antero-posterior skiagrams. (Plate II.) The base of the wedge

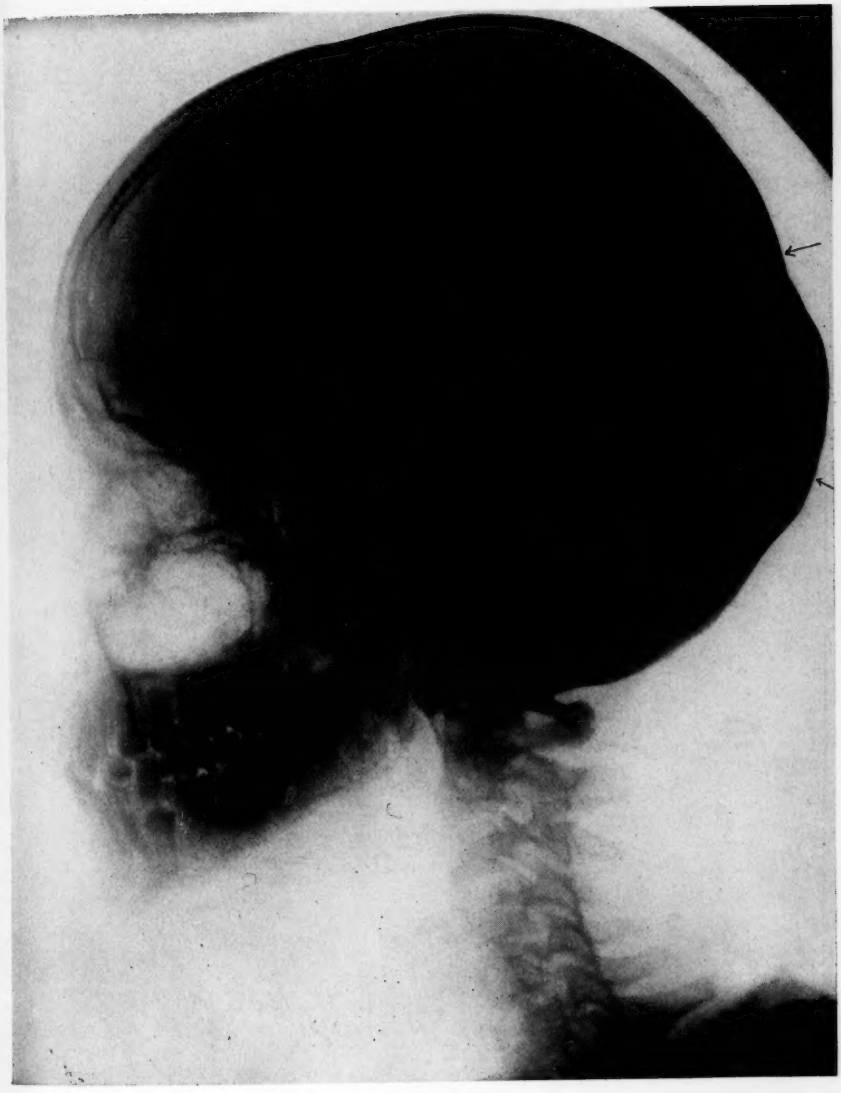

PLATE 1.

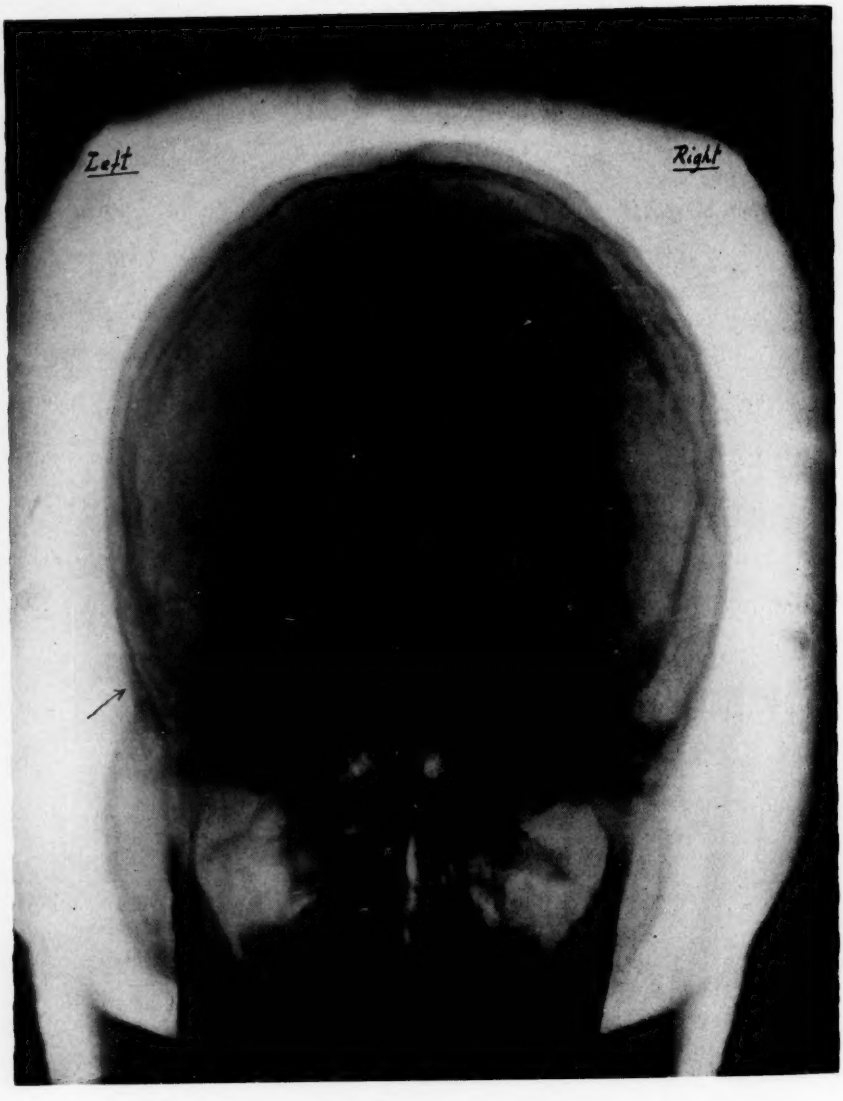

PLATE 2.

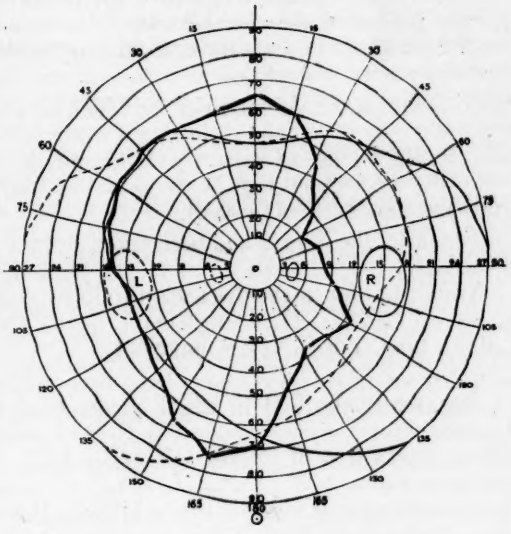

FIG. 1.

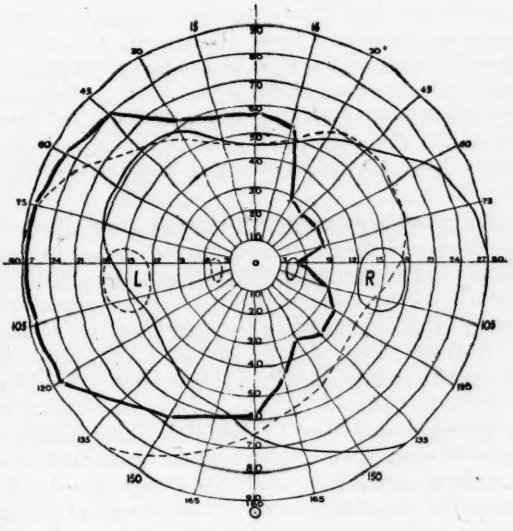

FIG. 2.

is at the occipital pole, immediately above the tentorium and is therefore closely related to the visual cortex. I am told by radiologists that the vessel walls must have undergone calcification in order to produce so dense a shadow.

With regard to the particular enquiries for which he was brought to me, I pointed out first that the right eye was already involved, and secondly I said I saw no reason why he should not go to school in the ordinary way but that he should be entirely debarred from vigorous exertion, such as football, etc.

At a later date, after the X-rays had been taken and the diagnosis had thus been completed, he was seen by Dr. C. P. Symonds, who has made reference to him in the discussion following Dr. Parkes Weber's paper on Cerebral Haemangiomata, in the *Proc. of the Roy. Soc. Med.*, 1929, Vol. XXII, 4, page 431.

A number of cases of this kind have been reported by various observers. Apparently the first in which evidence of a calcified meningeal haemangioma was recognised by X-ray examination on the living patient was that of Parkes Weber, in 1922. (*Jour. of Neur. and Psychopath.*, 1922, Vol. III, 134.) Previous to this they had been discovered at autopsy; thus D'Arcy Power reports a case (*Trans. Path. Soc.*, 1888, XXXIX, 4.) in which the mass of vessels extended deeply into the brain substance, and Drysdale (*Ibid.* 1904, IV, 60) reported two cases, also with autopsies, in which a similar condition was present. Since that time a number of cases of such tumours involving different parts of the brain and indeed the spinal cord have been discovered, and have often been associated with naevi of the skin of the face, having a trigeminal distribution, or in the case of the spinal cord, a distribution along the corresponding spinal nerve. These naevi, when present, often serve to point the diagnosis, as in the present case, but the intracranial naevi do not always have any such external indication as to their nature.

These meningeal haemangiomata are of very special interest to ophthalmic surgeons from several points of view. In the first place is their association with congenital malformations of the eyes themselves. Of these the most striking is the frequent presence of buphthalmos. It was present in three of Cushing's cases (Harvey Cushing, *Jl. Amer. Med. Assoc.*, 1906, XLVII, 178), two cases of Brushfield and Wyatt (*Brit. Jl. of Child. Dis.*, 1914, XI, 25), and two cases of T. R. Aynsley (*Brit. Jl. of Child. Dis.*, 1928, XXV, 197), and one case of Parkes Weber (*Loc. cit.*, 1922). In Parkes Weber's case the buphthalmic eye became blind and ulcerated, and was afterwards removed by Dr. C. Markus, and it is of interest to observe that Dr. Markus found no evidence of an intraocular naevus.

Another ocular abnormality is heterochromia iridis. This has been referred to by Parkes Weber who states that it was present in his own case, in one of Brushfield and Wyatt's, and in Sheldon's case. I have no note of it in my present case, but I have had the opportunity of examining a case of Mr. L. B. Rawling's which at present is not reported, and in which the iris on the side of the haemangioma was more heavily pigmented than on the opposite side. The difference in the colour of the iris, unless it is well marked, is readily overlooked, and this is particularly likely to happen if the irides are dark in colour, or perhaps the difference is observed but is looked upon as of no interest. In the case to which I have just referred the heterochromia might readily have escaped detection except for the fact that one suspected it might be present, and I think it likely that were all cases examined especially carefully with regard to this point it would prove to be common.

In other cases other mild malformations occur which perhaps do not easily obtrude themselves. In the present case it had been suggested that there was partial optic atrophy in the left eye and that this fitted in with the defective sight of which the boy complained. The apparent pallor of the disc, however, was due to the presence of a small congenital coloboma, which was not present in the right eye. There was a slight degree of congenital displacement of the lenses of both eyes.

In Mr. Rawling's case referred to above, the eye on the side of the lesion had a high degree of mixed astigmatism, whereas the opposite one was normal.

Lastly there is the important question of the involvement of the optic radiations and the visual cortex by the tumours giving rise to homonymous defects in the fields of vision. This is well exemplified in the present case. In one of Mr. Rawling's cases, which I have examined, and to which I have referred above, in which he found at operation a large naevus over the parietal region of the right side of the brain, there was, as one would expect, no defect in the visual fields, but in his other case, which I have not at present had an opportunity of seeing, it would seem exceedingly likely from the appearance of the skiagrams, and from the great similarity to my own case that a defect of the field of vision would be found.

It will be noticed as a striking fact in all the above instances, that the abnormality of the eye, whether it is buphthalmos, heterochromia iridis, coloboma of the disc, or a high refractive error, was on the same side as the tumour of the brain, the only exception being the dislocation of the lens which was present in both eyes.

Having regard to the frequency with which buphthalmos has been found to be associated with intracranial haemangioma, it would seem worth while to have a skiagram taken of the skull in

any case of buphthalmos, on the chance of intracranial haemangioma being present. I have had one case of double buphthalmos examined in this way since commencing this paper and in it no tumour was shown by X-ray.* I suggest too that a more critical examination of the eyes in these cases is likely to reveal a greater incidence of the defects referred to above than has been found in the past.

The association of retinal haemangiomatosis, Coats' disease (v. Hippel's disease), with haemangioma of the cerebellum and sometimes other parts of the body has been pointed out by Lindau, and Parkes Weber makes the suggestion that the scattered cases of Coats' disease which have been reported in this country should be re-examined with regard to the possible occurrence of a cerebellar haemangioma.

I published in 1914 (*Roy. Lon. Ophthal. Hosp. Reports,* 1914, XIX, Part III, 411) the first case of a melanoma of the choroid which had been examined ophthalmoscopically during life and was afterwards obtained for pathological investigation owing to the death of the patient from myasthenia gravis, and in the first volume of this Journal I published a second case in which I obtained the specimen for pathological examination owing to the death of the patient from a cerebral tumour. de Schweinitz and Shumway (*Trans. Amer. Ophthal. Soc.* 1905, X, 439) published what seemed to be a similar case, which was discovered post mortem in a patient who also had died of a cerebral tumour.

Melanomata of the choroid are not very common, but are not exceedingly rare. They do not undergo change. I have recently ascertained that the patient from whom the coloured drawing in the Moorfields Reports referred to above was made, is quite unchanged after fifteen years. At the same time, having regard to the above mentioned cases, and to the group with which we are now dealing, I repeat the suggestion that I made when showing the above case at a meeting of the Ophthalmological Society some ten years ago that the possible association of choroidal melanomata with intracranial tumours should be watched for.

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*I have since had another case of my own examined and Miss Ida Mann has been good enough to allow me to have a case of her own examined, and in both cases the result was negative.

ANNOTATIONS

Drawings of the Fundus

The art of pictorial representation of diseased conditions of the fundus dates of course from the year 1851, when Helmholtz gave the ophthalmoscope to the world. The value of these pictures for teaching purposes has long been recognised and from time to time atlases of the fundus have been published; one of the earliest of these was Liebreich's atlas, published in 1863. For many years this held the field, and it is still an extremely good atlas. In 1870 de Wecker and Jaeger brought out their great French atlas; this is now a scarce and valuable book. Frost's *Fundus Oculi* (1896) was the next milestone; it is a monument of Mr. Head's artistic ability, and we have always considered it as the best all-round atlas of the fundus. Parent's "Atlas d'Ophtalmoscopie" was published in Paris about 30 years ago. The fine plates in Gowers's Medical Ophthalmology deserve mention, and Haab's Atlas, which, from the student's point of view, is a cheap work, well within their means. In Germany, Adam's *Ophthalmoskopische Diagnostik* came out in 1912, and in the following year Oatman, in America, brought out his *Diagnostics of the Fundus Oculi.* A good English atlas at a reasonable price was that of Lindsay Johnson and Head, which was published in 1911.

At the present time in England practically the whole of this branch of ophthalmology is in the hands of Theodore Hamblin, Ltd. It is safe to say that the collection of drawings at 15, Wigmore Street is unique; certainly there is nothing approaching it for completeness in this country, and we much doubt if it can be equalled anywhere else in the world.

We feel that this excellent collection is not so widely known and made use of by ophthalmic surgeons and students as it might be. We have confined our remarks so far to fundus pictures, but Hamblin has an extensive collection of drawings of external diseases as well as lantern slides. As regards these latter it should be known to all that the Adams Frost collection of lantern slides of the drawings in the *Fundus Oculi* is housed in the Bowman Library and can be borrowed by applying to the Hon. Librarian.

The art of coloured pictures of external conditions has been practised for many years and no better atlas of such conditions can be imagined than Dalrymple's famous atlas of eye pathology.

In our advertisement columns of this month will be found more details of this department of Hamblin's enterprise. We would commend the drawing department at Wigmore Street to our readers. It is open between the hours of 9 a.m. and 6 p.m. Visitors are welcome to inspect the collection; most of the illustrations of

papers in our journal will be on view together with a series of drawings of similar conditions. The pictures are hung in special frames containing about half a dozen each in a good light. We would wish to express the thanks of the ophthalmic fraternity to Mr. Rayner Batten, who has spent much time in cataloguing and arranging the drawings in this department.

Inaccurate Bibliographies

To no one does the familiar injunction, "verify your references," apply with more force than to the worker in the scientific field. A paper on a scientific, or even a clinical subject loses much of its value if accompanied by a host of erroneous or unrecognisable references. In this connection it is sad to see the blunders which appear in some European journals, the editors of which appear to take little trouble to check the names and titles submitted by their contributors. We trust that our readers will not jump to the conclusion that THE BRITISH JOURNAL OF OPHTHALMOLOGY is immune from error; "*humanum est errare*," and we plead guilty to making occasional mistakes. It is in the matter of foreign names that the chief source of error usually lies, and, while it is the duty of the author of a paper to see that the names are correctly spelt in the manuscript (which in the author's own interest should be typewritten), it is incumbent on the editor to correct obvious misprints. It is not fair to the compositor who has to set the type to expect him to be able to read foreign names in the hand-writing of most authors.

These reflections are the outcome of musings on the translation into French of some of the recent work of Stewart Duke-Elder (a serious pitfall these double-barrelled names), every page being headed with the name Stewart. The same journal translates "under the chairmanship of the late Sir Wm. M. Bayliss" as "sous la présidence de feu Sir Wim. M. Baylin." Similarly McCrae becomes Macrac, Orr is Orn, Cramers is Kramer, while Colebrook, Eidinow, and Hill appear as Colebrock, Eidinowd, and Holl, with Hell as an occasional happy variation. The fact that the mistake in the author's name is corrected in the following issue, without apology or explanatory remark, in no way diminishes the confusion which must be felt by the literary searchers.

Another serious inaccuracy in certain foreign journals is to be found in prescriptions of drugs of an unusual type. Any reader who acted on the advice given without verifying the dosage might have serious cause to regret his confidence.

We would plead with the editors of such journals that inaccuracies of the type which we have mentioned are good neither for their reputation nor for science in general.

ABSTRACTS

I.—OPTICS AND REFRACTION

(1) **Fincham, E. F. (London).—The function of the lens capsule in the accommodation of the eye.** *Trans. of the Optical Soc.*, Vol. XXX, 1928-9.

(1) In 1924 **Fincham** published (*Trans. Optic. Soc.*, Vol. XXVI, No. 5, 1924-5, extracted in the *Brit. Jl. of Ophth.*, Vol. IX p. 524, 1925), a paper on the changes in the form of the crystalline lens in accommodation, and drew attention to the varying thickness of the capsule of the lens and its probable action in producing the well-known " lenticonus," discovered by Tscherning, which occurs at the anterior surface of the lens during accommodation. Here it was suggested that thicker and stronger parts of the capsule will resist distension and so cause the surface to be flattened, whereas bulging will take place where the capsule is thinner; consequently, during accommodation the tension upon the surface is relaxed and the capsule becomes distended by the pressure of the lens substance, the distension being most marked where the capsule is weakest.

The author now returns to the subject and, in an able and interesting paper, continues the discussion.

Method of Investigation.—Nearly all the observations were carried out on the lenses of animals, and so it was not possible to correlate the form of the lens during accommodation in life with the curves of the surfaces of the lens when removed from the eye. The aim was to record the characteristic contour of the surface of the lens when held by its normal suspensions and also when allowed to assume its free shape.

The method of making these records was to photograph the image of a target as reflected in the surface. The target was of peculiar design so that its image produced by a convex spherical surface was square. The target was fixed at a constant distance from the surface examined and the camera directed directly downwards.

The following observations were made:—

1. The cornea and iris were removed, the eye supported in a cup-shaped vessel and the lens bathed in normal saline solution. The image of the target in the anterior surface of the lens was photographed after excess of saline solution had been removed.

2. Similar photographs were taken after the following dissections:—

   (*a*) The posterior half of the eye was removed and the cut surface of the vitreous supported by a flat surface.

   (*b*) The zonule and vitreous connexions of the lens were severed and the lens left lying on the vitreous.

   (*c*) The lens was removed and placed in a little cup of plasticine approximately the same curvature as the posterior surface of the lens.

The eyes examined were from the pig, sheep, reindeer, rabbit, monkey (four species), baboon (two species) and one human eye from a patient of seventy years.

The Form of the Lens when removed from its Suspensions.—It is assumed that the influence of the capsule upon the form of the lens will be greatest when it is unflattened by the tension of the zonule; and according to the hypothesis of Helmholtz in a state of maximum accommodation.

It was found that, in animals lower than the primates, the anterior surface of the lens was approximately spherical, whilst in baboons and monkeys it had the characteristic conoidal form of the accommodated human lens. The image from the lens of a human subject aged 70 years showed marked barrel distortion, indicating that the surface was more steeply curved in the peripheral zones. This lens was quite rigid, and the radius of curvature of the central part of the anterior surface 9.4mm., quite a normal value, in the unaccommodated eye. This also proves that the reduction in amplitude of accommodation is due to sclerosis of the lens.

The Effect of Alteration of Tension upon the Lens.—In the lower animals the images were very similar before and after the lens had been freed from its connexions with the suspensory ligament. In the baboons and monkeys, on the other hand, there was a remarkable difference in the two conditions. The anterior surface of the lens was noticeably flatter when held in suspension; when released not only did the lens increase in curvature in its centre, but the conoidal form of the surface became more marked.

It is noted that no actual proof exists that the freed lens is in the state of maximum accommodation, although this has been assumed.

The Thickness of the Lens Capsule.—In the lower mammalia the capsule was approximately uniform in thickness over the anterior surface, and in each case was quite thick at its centre. The lens capsule of monkeys showed the same general type of variation in thickness as is found in the human capsule.

The Effect of Removing the Capsule.—In order to test the effect of the capsule upon the form of the lens photographs in profile were taken with and without the capsule. The lens was removed from the capsule intact (as proved by subsequent sections of the capsule). The soft lens was immersed in castor oil so as to prevent distortion. In all cases the soft lens removed from the capsule became flattened, and it was also noted that the lens substance could be easily distorted by pressure with a hard object and always returned to the same form.

CONCLUSIONS

1. The lens surface increases in curvature, and assumes the accommodated form, when released from its suspensions.

2. The conoidal form of lens surface is found only in those cases where the anterior capsule has a relatively thin central area surrounded by a zone of greater thickness.

3. The primate lens loses its characteristic accommodated form when the capsule is removed.

4. The amplitude of accommodation is small in those animals which have an anterior capsule of uniform thickness.

The Value of the Conoidal Surface in Accommodation.—A few observations have been made to show that the pole of the lens moves forward 0.25mm. to 0.50mm. during accommodation, whereas the equatorial diameter is only reduced by 0.50mm. and the peripheral parts of the surface remain stationary. For an accommodation of 8D. the radius of the curvature of the central part of the anterior surface of the lens changed from 12mm. to 5.5mm. If the whole surface of the lens were affected in this way it would need a much greater reduction in the equatorial diameter of the lens. It is thus seen how advantageous is the conoidal deformity produced by accommodation.

The Capsular Theory of the Mechanism of Accommodation.—The change which the form of the lens undergoes when the capsule is removed appears to indicate that the effect of the capsule is to compress the lens substance around the equator and in the zones of greatest thickness. Such a compression would cause the central bulging of the anterior surface which is typical of the accommodated lens.

The lens removed from its capsule appears to be in the unaccommodated state. May we not infer that when an eye is not accommodated the compressing force of the capsule is neutralized or held off by the traction of the zonule? The process of accommodation would then consist of the contraction of the ciliary muscle releasing the tension from the zonule, which in its turn releases the capsule

and allows it to mould the lens into the accommodated form. This power will necessarily decrease as the lens grows harder and more resistent in later life.

According to the Helmholtz hypothesis the accommodated condition of the lens is the natural form of the lens, and the lens remains in its unaccommodated form by the constant traction of the suspensory ligament, yet we know that the lens grows harder in later life and should therefore produce myopia by being fixed in the accommodated condition, which is not the case. Apart from this it has seemed unnatural that the lens in the unaccommodated condition should not be in a state of rest.

Fincham suggests that the lens substance (which is less readily distorted in later life), is in its natural form when unaccommodated and that the process of accommodation consists of the contraction of the ciliary muscle releasing the tension of the zonule which in its turn releases the capsule and allows it to mould the lens into its accommodated form.

All credit is due to Fincham for showing us that the peculiar conoidal form assumed by the lenses of primates during accommodation, is wholly explained by a relaxation of the zonular fibres owing to the special thinning of the central part of the capsule that is found in them. The relaxed primate lens loses its characteristic form when the capsule is removed.

Unfortunately Fincham has been unable to obtain any young human eyes. If any reader could send an uninjured young eye in normal saline to E. F. Fincham, Northampton Polytechnic Institute, London, in order to complete his work, I am sure he will be most grateful.

I have abstracted this paper at some length not only because of the ingenuity of the investigation, but because this paper (together with that published in 1925) seems to me to place the Helmholtz hypothesis beyond doubt.

CHARLES GOULDEN.

(2) **Essed and Soewerno.—Experimental myopia in apes. (Ueber Experimentalmyope bei Affen.)** *Klin. Monatsbl. f. Augenheilk.*, Bd. LXXX, S. 56, 1928.

(2) **Essed and Soewerno** have performed a series of experiments on apes which tend to confirm, in the authors' opinion, Levinsohn's theory of the aetiology of myopia. It will be remembered that in this writer's opinion one of the most important factors in the aetiology of myopia is the action of gravity upon the globe of the eye. The authors took six apes and placed them in boxes, so that their heads hung down from a hole in the side, with an arrangement by which they were prevented from looking up.

They were confined there for periods of several months. Two showed little or no change, one after six months and the other after two months confinement. In the others the original hypermetropia turned into a myopia, the myopic change being from 1 to 1.5D. Controls showed negative results.

W. S. DUKE-ELDER.

(3) **Frenkel, Henri** (Toulouse).—**Unilateral myopia.** (**La Myopie monolatérale.**) *Arch. d'Ophtal.*, April, 1928.

(3) **Frenkel** is of opinion that unilateral myopia is more common than is generally known; he refers to the not infrequent discovery of it in applicants for pension or compensation who have been quite unaware of the difference between the two eyes until they come up for examination. His paper is largely statistical, with tabular arrangement of figures obtained from hospital and other clinical sources. In the tables he shows the numbers of cases of symmetrical myopia, of myopia with unilateral predominance, and of purely unilateral myopia. The relative numbers in the two sexes and at ages from 5 to 70 years are also given. Unilateral myopia appears to be more frequent in females than in males and on the right than on the left side.

In reference to the question of heredity the author states that while it is easy to show that myopia is hereditary it is difficult to prove inheritance in the unilateral myopia: he gives notes of a few illustrative cases.

In his concluding remarks Frenkel discusses the question of correction of refractive errors, in high degrees of anisometropia; he notes that, if correction is begun at an early age the subjects of unilateral myopia show a remarkable tolerance of correcting lenses differing by as much as 4 to 6 dioptres.

A bibliography containing 19 references is appended.

J. B. LAWFORD.

(4) **Incze, A.** (Budapest).—**The constitutional nature of myopia.** (**Ueber die Myopie als eine konstitutionale Veränderung.**) *Zeitschr. f. Augenheilk.*, Vol. LXVII, p. 20, 1929.

(4) In an investigation into the physical type of 188 myopes, **Incze** found that 72 per cent. (136 cases) were of the asthenic type. He considers this no coincidence, but regards myopia as an expression of generalised weakness of connective tissue throughout the body—holding this view in spite of the fact that in earlier investigation as to the refractive condition of asthenic people, he found that myopia was present in only 13 per cent. of asthenics. As supporting the view of a constitutional basis of myopia, the

author quotes the findings of other observers to the effect that myopia is now frequent in thin, pale subjects, that it often occurs in flat-footed individuals, and that the " curve of myopes runs parallel to the mortality curve of tuberculosis."

A. SOURASKY.

(5) **Birnbaum, H. (Antwerp). "School myopia." (La "myopie Scolaire." Existe-t-elle?)** *Arch. d'Ophtal.*, January, 1929.

(5) **Birnbaum** has written a long reasoned article, which would lose much of its force in an abstract. The term " school myopia " he interprets as " myopia acquired at school, as a consequence of school work." In his opinion the widespread belief in school myopia has no reliable foundation and he attacks it vigorously. His facts, statistics and deductions are well arranged and form interesting reading. His personal view is that " the evolution of myopia and of refraction of the eye generally should be considered as ontogenetic; an evolution determined in the embryological stage, as is true of the form and colour of the moustache which, also developing long after birth, is yet determined in the germ cell"

J. B. LAWFORD.

(6) **Lancaster, Walter B.—The Ames spectacle device for the treatment of cyclophoria, with a report of a successful case.** *Arch. of Ophthal.*, July, 1928.

(6) **Lancaster's** article concerns a case of cyclophoria in a well-to-do professional man aged 47 years, who was unable to read for more than 10 minutes at a time, although he wore an adequate correction for his error of refraction. Tests with a Maddox rod, vertical in front of one eye and horizontal in front of the other, showed about 2° of positive cyclophoria. This apparently did not trouble him in distant vision, but made reading intolerable. The patient would not consent to read with one eye covered, so he was referred to Professor Ames, who devised an exceedingly ingenious apparatus which gave him relief. It is based on the following observation:—

" Hold a hand-mirror vertically before you, but at an angle of about 45° with the line of vision and a few inches from the eye . . . then vertical or horizontal lines on the wall behind you will appear vertical or horizontal in the mirror. Now tilt the mirror so that it is no longer vertical, the image is moved up or down and the vertical or horizontal lines are tilted. By interposing a second mirror parallel with and facing the first, it is possible to make the eye see the image of objects in front of it instead of behind it and, by putting in a prism, to correct the upward or downward displace-

ment produced by the tilting of the mirror. One more correction has still to be made if the apparatus is to be used for reading, because the image as seen by the eye using the mirrors is noticeably smaller than that seen by the other eye, owing to the longer path of the rays. This is overcome by interposing two lenses whose effect, figuratively speaking, is to carry the nodal point of the eye forward. The whole apparatus is easily accommodated in one side of a spectacle frame, and screws are provided so that the required degree of torsion in the image may be maintained. The patient for whom the instrument was made obtained complete relief from the first day, and has now been using it for over a year with the greatest satisfaction.

F. A. WILLIAMSON-NOBLE.

(7) **Morsman, L. W. (Hibbing, Minnesota).—Velonoskiascopy.** *Amer. Jl. of Ophthal.*, June, 1928.

(7) Velonoskiascopy is a subjective test based on the following observation. If an ametropic eye views a vertical white line against a black background, the line appears blurred. If a vertical thread be now held in front of the eye, a central black interval will appear in the image, the width of the interval varying with the amount of ametropia. In cases of astigmatism, a cross with the lines set at the principal axes of the astigmatism may be employed instead of a single line, and cross wires may be held in front of the eye, the wires being parallel with the arms of the cross. Black intervals are now seen in both arms of the cross, and if astigmatism be present, one black interval will be wider than the other. The astigmatism may then be estimated by the strength of cylinder required to make the two black intervals of equal width. In cases where the position of the cross wires and the cross does not correspond with the axes of the astigmatism, the black interval appears to cut obliquely across the white line instead of bisecting it. In practice a wall chart consisting of a circular disc of red cardboard 49cm. in diameter is employed; it bears a cross consisting of two strips of white paper 4mm. wide and 38.5cm. long. On the other side is a series of radiating white lines, placed at 30° intervals and used to determine the axes of the astigmatism. When this has been found, the chart is reversed and hung in the appropriate position. In the trial frame there is placed a black disc, with a central hole 6mm. in diameter, occupied by two cross wires 0.5 or 1.0mm wide, the lines of wires being made to coincide with the arms of the cross and to intersect at the centre of the pupil. Morsman advises the performance of a preliminary retinoscopy to give an idea of the amount of error. This is then overcorrected with spheres so that the patient is made 1 or 1.5D. myopic in the major meridian, and

he is told what to look for. Minus cylinders are now introduced with their axes along the line of the broader interval until equality is reached. The fogging sphere is then reduced, note being made whether the dark areas remain equal in size all the time. The test calls for the exercise of considerable powers of observation on the part of the patient, and this, of course, is its chief drawback.

F. A. WILLIAMSON-NOBLE.

II.—GENERAL MEDICINE

(1) **Meisirow, M. E. (Chicago). — Central choroiditis due to toxaemias of pregnancy.** *Amer. Jl. of Ophthal.*, May, 1927.

(1) **Meisirow's** case was that of a woman, 3 months pregnant, who had lost 20 lbs. in two months, and was complaining of backache, headache and dimness of vision which began at the second month of her pregnancy. The ocular findings were: R.E. disc blurred, vessels narrow, fresh exudate 2 mm. wide at the macula, involving retina and choroid. L.E. thin haze in lower part of cornea, disc blurred, fundus otherwise normal. Two days later the central exudate had increased in size, and about the sixth day pigment was developed in the papillo-macular bundle, *i.e.*, between the disc and macula. The eye condition became worse in spite of eliminative treatment during the next fortnight, so the uterus was emptied. Progress of the disease was arrested and vision gradually improved from perception of light to 4/10 in each eye. Four months later, an irregular white area was found about 2/3 D.D. just below the macula with some pigment at its upper border —remnants of the ribbon-like pigmentary change which had developed in the papillo-macular bundle. It is notable that there was no sign of renal involvement in this case. The author concludes his article by quoting Zentmayer's rule: " If retinitis develops before the sixth month, pregnancy should be terminated. If at the eighth month, carry to full term. Between the sixth and eighth month be guided by the visual disturbance. If vision is poor, terminate pregnancy."

F. A. WILLIAMSON-NOBLE.

(2) **Hebert, Dr. (St. Quentin).—Two cases of neuro-retinitis in the course of pregnancy. (Deux cas de neurorétinite au cours de la grossesse).** *La Clin. Ophtal.*, February, 1928.

(2) It is difficult to know how to describe the two cases related **by Hebert.** Both were cases of distinct neuro-retinitis, both

occurred in the course of pregnancy, both had arterial hypertension, both showed only traces of albumen in the urine, both were such as to lead to induction of labour. Yet the one presented retinal haemorrhages, while the other did not. The case with no retinal haemorrhages proved to be a typical case of retinitis of pregnancy and recovered; the other had a syphilitic basis and, after a partial recovery following delivery, died totally blind a year later. The author draws no decisive conclusions and admits that the cases do not help us much to understand a condition "of which the precise aetiology still escapes us."

ERNEST THOMSON.

(3) **Krauss, Frederick, and Spikes, N. O. (Philadelphia, Pa.)—Oedematous anthrax of the face resulting in meningitis.** *Amer. Jl. of Ophthal.*, May, 1926.

(3) **Krauss** and **Spikes'** case is of interest, particularly in showing that a stye may not always be what it seems. The patient, a piercer of yarns, had noticed a small swelling on the right upper lid for two days. It increased enormously, in spite of treatment, and the side of the face became swollen. A deep incision made in the upper lid produced only a blood-stained discharge and cultures were negative. The swelling became hard and brawny, and the edges of the wound somewhat necrotic.

Cultures now showed a few cocci. The condition rapidly became worse, Cheyne-Stokes respiration developed, the neck became rigid, and the hands and fingers athetoid. The cerebro-spinal fluid was found to contain cocci and anthrax bacilli. Pulmonary oedema then developed rapidly and the patient died within thirty-six hours of admission to hospital. Post-mortem there was found a suppurative meningitis due to anthrax with extensive oedema and haemorrhage in all the deeper structures. A feature of the case was marked absence of pain, in spite of the swelling and inflammatory changes.

F. A. WILLIAMSON-NOBLE.

(4) **Constans, George M. (Bismarck, North Dakota).—Ocular pemphigus.** *Amer. Jl. of Ophthal.*, November, 1927.

(4) **Constans'** paper opens with a short review of the literature on pemphigus, in which it is pointed out that prior to Cooper's description of the disease in 1858, ocular pemphigus was unknown as an entity. The cause is unknown and the fluid in the blebs has been found to be sterile. Four types of pemphigus are described: (1) Acute, which is often fatal, runs a rapid course and is associated with severe general symptoms; (2) Chronic, in which there may be successive crops of blebs; (3) Vegetant, in which the bullae

become converted into excoriations in which vegetations grow; (4) Foliate, which is gradually progressive and almost always fatal, the lesion affecting the skin and mucous membranes, and healing slowly. In ocular pemphigus, the symptoms comprise xerosis, the formation of blebs, associated usually with itching and burning of the eyes. The condition is usually bilateral and, in a late stage, marked shrinkage of the conjunctiva occurs with dryness of the cornea and loss of vision. The treatment is merely symptomatic and comprises the use of a bland lotion and oil. Three cases are described:—The first occurred in a married woman, aged 53 years, who noticed itching of the eyes followed by swelling of the lids and severe chills in July, 1925. The temperature rose to 104° F. and there were general manifestations of pemphigus. The skin exfoliated from the lids and a greyish exudate formed over the palpebral conjunctiva. The general condition improved, but the cornea became involved in the middle of August and showed sloughing of the superficial layers which went on to perforation and formation of hypopyon ulcers. In October the conjunctiva was still injected, the right cornea was hazy and showed facets, while the left was nearly normal, except for a central nebula from the old ulcer. In November, the conjunctiva was found to be replaced by milky-white new connective tissue, and there were numerous adhesions between the globe and lid. In the second case there was general pemphigus with swelling of the lids, but no corneal involvement or conjunctival adhesions. The third case was one of primary pemphigus of the eye. The palpebral conjunctiva of both upper lids showed irregular scars with red areas of denuded epithelium and there were adhesions. The corneae were clear. Nine months later there was scarring and vascularization of the left cornea, and seven months later still, small "mutton fat" deposits were found in the substantia propria.

F. A. WILLIAMSON-NOBLE.

(5) **Stein.—Thrombo-phlebitis of the opposite orbit and crossed paralysis of the abducens in otitis media. Statistics of the eye symptoms in otitis media and its complications. (Thrombophlebitis der gegenseitigen Orbita und gekreuzte Abduzensparese bei Otitis media. Statistisches über Augensymptome bei Otitis media und ihren Komplikationen.)** *Klin. Monatsbl. f. Augenheilk.*, Bd. LXXX, S. 22, 1928.

(5) The case described by **Stein** was that of a boy, aged 15 years, who had chronic otitis media in the left ear. An acute exacerbation developed in which a paralysis of the sixth nerve became evident on the opposite side. The post-mortem examination showed that he had a thrombo-phlebitis of the cerebral sinuses

which started in the left sigmoid sinus, and travelled up to the left cavernous sinus, through the circular sinus, to the right cavernous sinus. From there it travelled to the orbital veins and produced an orbital cellulitis on the right side, while, on the left side, the thrombosis was limited to the cavernous sinus and caused no apparent symptoms.

Stein takes the opportunity of reviewing the ocular complications, which were met with in 850 cases, of suppurative otitis media. Of these 795 had no cerebral complications, while 55 had.

Otitis media without cerebral complications—795 cases.

Nil in eyes—724 cases (91.1 per cent.)
Hyperaemia of disc—bilateral 31 cases (4.1 per cent.)
homolateral 25 cases (3.0 per cent.)
contralateral 2 cases (0.2 per cent.)

Optic neuritis—bilateral 7 cases (0.9 per cent.)
homolateral 6 cases (0.7 per cent.)

Otitis media with cerebral complications—55 cases.

Nil in eyes—36 cases (65 per cent.)
Hyperaemia of disc—bilateral 8 cases (14.5 per cent.)
homolateral 7 cases (12.5 per cent.)
Optic neuritis—bilateral 4 cases (8 per cent.)

W. S. DUKE-ELDER.

(6) **Ramsay, A. Maitland (St. Andrews).—Some ocular manifestations of cardiovascular disease, excluding renal complications.** *Practitioner*, September, 1928.

(6) In this useful article for general practitioners by **Maitland Ramsay** a few points may be picked out which the ophthalmic surgeon sometimes forgets.

Persistent autotoxaemia is always accompanied by a state of increased blood-pressure. Hyperpiesis therefore must be regarded as a symptom rather than as a disease. Hyperpiesis always is followed sooner or later by sclerosis of the walls of the arteries. Marcus Gunn has pointed out that old age does not of itself cause arteriosclerosis, but that it is always secondary to some cause capable of inducing and maintaining high blood-pressure. Sclerosis of an arteriole causes starvation of the capillaries which it feeds, and in consequence the retina suffers from want of nourishment. In these circumstances the walls of the capillaries soon become damaged, and much of the subsequent trouble can be traced to this cause. In the case of the retina, impaired nutrition

causes defective vision, which is indicated in the first instance by disturbance of the light sense, accompanied by disordered perception of colour. Capillary diapedesis, haemorrhage and oedema are the first milestones on the road ending in destruction of tissue. In the retina the whole pathological process culminates in atrophy of the optic nerve.

Although the occurrence of intraocular haemorrhage makes little difference to the prognosis with regard to life, yet it greatly increases the danger to sight. Obviously the amount of damage depends upon the size and the site of the haemorrhage. Small scattered flame-like extravasations in the nerve-fibre layer of the retina are, as a rule, never alarming, and unless they are very numerous the patient may be unaware of their existence until they are revealed by ophthalmoscopic examination. They usually become absorbed, but unless the underlying toxaemia can be eliminated or controlled, they are almost certain to recur, consequently crops of haemorrhages in different stages of absorption are frequently seen in the fundus over a period of months or years. On the other hand, when instead of being striate or flame-shaped the blood clots are more or less circular in outline and situated deeply in the retina or in the macula, the regular mosaic of rods and cones will be damaged and sight seriously and permanently impaired. Retinal haemorrhages are largely of the nature of capillary leakages and therefore differ from cerebral haemorrhages, which occur when an arteriole with walls weakened by arteriosclerotic changes bursts.

Mott has pointed out that advanced arteriosclerosis with normal blood-pressure may exist in the retina as it does in the brain. In such circumstances the independence of the capillaries must never be overlooked, because although systolic blood-pressure may be below normal, capillary pressure may be much above normal. When structural changes predominate in the retinal vessels and there is deficient capillary circulation, caution must be observed lest strenuous attempts to lower a high arterial pressure impair still further the already inadequate capillary circulation. A sudden fall in blood-pressure is likely to precipitate the occurrence of retinal haemorrhage, which in these circumstances is frequently the precursor of a general breakdown in health.

A. F. MACCALLAN.

(7) **MacGillivray, Allister M. (Dundee).—A case of "Eales's Disease." (Recurrent intraocular haemorrhages).** *Lancet*, March 31, 1928.

(7) In two columns of the *Lancet* **MacGillivray** ably summarises the various causes which have been put forward to

explain this mysterious disease, and relates a tragic case of his own. The patient was a man aged 28 years, first seen on May 20, 1924. Up to the previous day he had had perfect eyesight. Quite suddenly a dense cloud came over the left eye. The vitreous was found to be filled with blood obscuring any view of the fundus. The vision was only P.L., that of the right eye being 6/6. The patient was not a bleeder and knew of none such in his family. His own doctor reported nothing in the general condition to account for the haemorrhage. Potassium iodide was given and under this the haemorrhage began to clear up, only to recur as badly as ever seven weeks after the first attack. Once again nothing was found in the general condition, but the blood coagulation time was found to be slightly lowered. Calcium lactate was substituted for the iodide. Six months later, there having been no further intraocular bleeding but several slight attacks of epistaxis having occurred, a clear view of the fundus was obtained. The V.A. was 6/60. " A narrow sickle-shaped band of white fibrous tissue was seen arising from and obscuring the optic disc. This fibrous band extended towards the macular region and projected some distance into the vitreous. The macula itself was not involved, the band curving below it to end in a fine point some two discs' breadth beyond. Several newly-formed blood-vessels were to be seen curling irregularly over the white mass and projecting into the vitreous." The author explains the formation of fibrous tissue as coming from the mesoblastic tissue of the blood-vessels, such fibrous tissue formation occurring in Eales's disease though not, as a rule, in vitreous haemorrhages of other types. By cicatricial contraction the bands may drag upon the retina, with the result that the retina becomes detached. This is what occurred in the author's case. Relapses occurred, and by December, 1925, a large blood-stained detachment of the retina could be made out. The patient was not seen again until February 8, 1928, when similar conditions, though not detachment, were found in the right eye, these apparently dating from an attack of sudden blindness a month previously.

The author makes the interesting statement that in May, 1926, the patient was admitted to hospital under W. E. Foggie for special investigation as to the nervous system. The following is a transcription of the note :—" He was found to have retention of touch in the right arm, the right side of the trunk, and the right leg, but analgesia and thermo-anaesthesia over the same area. There was some stiffness and weakness of the left leg. With the knowledge of recurrent intraocular haemorrhages in the left eye the condition was diagnosed as a haemorrhage into the spinal cord in the lower cervical region—a haematomyelia, with probably some pressure on the pyramidal tracts of the left side. The Wassermann reac-

tion was negative. The electrical reactions of the limbs were normal. The other systems were normal. Blood pressure 140/105. Blood coagulation time: 75 seconds—*i.e.*, lower than normal. The patient was very nervous and his general outlook gloomy." The author, naturally, gives a grave prognosis for, although actual detachment has not yet occurred in the right eye, this is likely to occur as in the left eye. Finally, it should be mentioned that at one period in the case injections of haemoplastin were tried but these failed to control the intraocular haemorrhagic relapses.

ERNEST THOMSON.

(8) **Clegg, J. Gray (Manchester).—The relation of ophthalmology with general medicine.** *Practitioner*, November, 1928.

(8) **Clegg** gives an interesting sketch of the relation of ophthalmology with general medicine. He mentions that interesting fundus condition, lipaemia retinalis, which occurs in young subjects and in which death almost invariably soon occurs. Foster Moore (*Trans. Ophthal. Soc. U.K.*, Vol. XL) recalls two cases from the literature on the subject in which recovery occurred and the fundi became normal in appearance. In a case under the care of Dr. Arnold Stott at Westminster Hospital which showed the typical appearances of lipaemia retinalis, complete return to a normal fundus condition occurred under dietetic and insulin treatment, and the young man is carrying on a normal life five months after he came under observation.

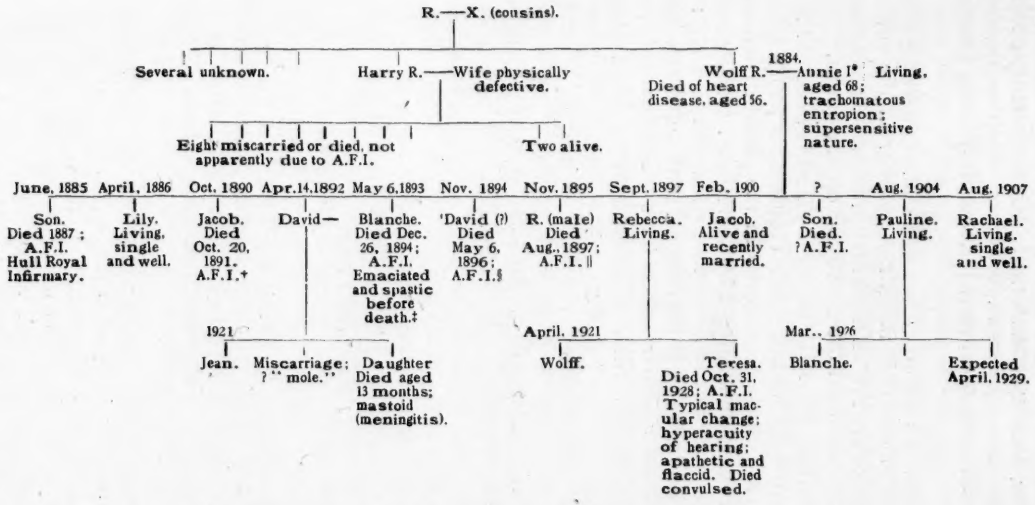
A. F. MACCALLAN.

(9) **Stenhouse-Stewart, D. D. (Hull).—Notes on a pedigree of amaurotic family idiocy (Tay-Sachs disease).** *Brit. Med. Jl.*, January 19, 1929.

(9) **Stenhouse-Stewart** records in this note a case of amaurotic family idiocy which proved on investigation to represent the second generation of the classical R—— family recorded by Kingdon of Nottingham. He states that his object in recording this case is twofold. First, the appearance of the disease in the second generation, and, if it should occur elsewhere in the family, the importance of determining if the heredity is solely through the female side. Secondly, what advice should be given to the remaining members of the family with regard to future children.

We reprint his genealogical table and references.

E. E. H.



* Other members of this family have no abnormal history so far as is known.

† Transactions of the Ophthalmological Society, Vol. X, p. 126. Quoted in the Transactions of the Royal Medical and Chirurgical Society of London, LXXX, 87.

‡ Transactions of the Ophthalmological Society, XIV, 129. Transactions of the Medico Chirurgical Society, LXXX, 87.

§ Transactions of the Royal Medical and Chirurgical Society of London, LXXX, 87.

‖ Ibid, Addendum.

(10) **Worms, G. (Paris) and Sourdille, G.—Ocular complications of enteropathies due to specific germs. Bacillus coli, enterococcus, bacillus dysenteriae. (Complications oculaires des entéropathies à germes spécifiques. Colibacille, entérocoque, bacille dysentérique.)** *Arch. d'Ophtal.*, December, 1928.

(10) This paper is a record of **Worms's** and **Sourdille's** study of three varieties of microbic intestinal disorder in the course of which ocular lesions are not infrequently present. An epidemic of bacillary dysentery afforded them unusual opportunities.

The causal relation of various forms of enteritis to ocular lesions has been so extensively studied since Trousseau's paper appeared in 1894, that there is little noteworthy in the present communication, apart from some interesting clinical records. In the bacillus coli group ocular complications are limited almost entirely to the tissues of the uveal tract and to the external tunics of the eyeball. In dysentery two forms of ocular lesions have been observed; scleroconjunctivitis and plastic iritis. Clinical notes of 12 cases under the writers' observation are given. Much less is known of the toxic effects of the enterococcus, and the references by Worms and Sourdille are taken from the work of Heitz-Boyer, who reported acute conjunctivitis (bilateral) in the late stage of enteritis, the enterococcus being identified in the faeces and urine.

J. B. LAWFORD.

III.—RETINA

(1) **Schmitz-Moormann, P. (Freiburg i.B.).—On the glycogen-content of the retina and its relations to the contraction of the cones. (Ueber den Glycogengehalt der Retina und seine Beziehungen zur Zapfencontraktion.)** *Arch. f. Ophthal.*, Vol. CXVIII, Pt. 3, 1927.

(1) The localisation of glycogen in the retina has been the subject of much diversity of opinion among writers, and the early researches of **Schmitz-Moormann** himself also showed very great variation in the situations where it was found, to such an extent that he concludes that this variability in its distribution can only be due to a post-mortem deposit of the glycogen and to faulty fixation.

By adopting a fresh method of fixation for frog's eyes he found that the glycogen was confined to the myoid, the contractile inner

segment of the cone, and, to a small extent, the adjoining cone stalk (*zapfenstiel*). With this carbohydrate reserve and its power of contraction the myoid possesses characters similar to those of muscle fibre. The presence of this glycogen-reserve makes it extremely probable that the change from chemical to kinetic energy on contraction of the myoid takes place in the myoid itself. The resulting chemical products of metabolism are to be regarded as the expression of this change of energy on the contraction of the myoid. The products that stimulate the retina on the action of light, so far as the cones are concerned, are furnished by the myoid itself.

He thus thinks it possible to bring the two opposing theories for the contraction of the cones, the chemical and actinic, into substantial agreement.

As regards the extension and contraction of the rods and the old question of their active or passive movement Schmitz-Moormann shows that they are entirely devoid of glycogen, and therefore have no contractile part similar to the myoid of the cones. Hence he concludes that their movement is essentially passive.

THOS. SNOWBALL.

(2) **Loddoni, Giovanni (Genoa).—Chorio-retinitis juxta-papillaris in a case of papilloedema after head injury. (Corioretinite juxta-papillare in papilla da stasi per trauma cranico.)** *Boll. d'Ocul.*, Vol. VII, No. 11, p. 1099, 1928.

(2) An interesting case of a girl, aged 19 years, who, several days after a fall, lost the sight of the *right* eye. In falling, she had received a blow on the *left* parietal region, resulting in a cut which, however, did not require to be stitched. There was no concussion and the patient was able to go on with her work for several days, though she felt heavy-headed and tired in the eyes. There was some pain on movement of the eyes which gradually increased. It was only on the 8th day that the sight of the right eye became badly affected and the headache became intense, but it was not till the 17th day that she came under observation. When first seen, right vision was reduced to finger counting at 40 cm. The visual field was reduced to 20° and there was paresis of the right internal rectus and right superior oblique. The disc showed definite papilloedema with haemorrhages (measurements not given). The left eye was normal and vision full. While under observation she developed a typical macular fan. The most interesting point, however, about the case was that she also developed on the nasal side of the disc an area of choroido-retinitis. Vision ultimately returned to full normal, but the visual field showed a large scotoma in the temporal area corresponding to the patch of choroido-

retinitis. X-rays revealed a small contrecoup fracture of the base involving the sphenoid.

The interesting features of the case are (1) the comparatively late development of papilloedema and visual loss in a case of traumatic haemorrhage into the nerve sheath, and (2) the development of a choroido-retinitis juxta-papillaris in association with a papilloedema.

LESLIE PATON.

(3) **Beach, Sylvester Judd (Portland, Maine).—Treatment of obstruction of the central retinal artery.** *Amer. Jl. of Opthal.*, May, 1927.

(3) **Beach** is inclined to regard this lesion as due to "endarteritis obliterans, often more or less complicated by spasm or thrombosis" rather than to embolism. He gives details of a case seen by him in 1916, which occurred in a woman aged 52 years. There was sudden and complete loss of vision in the left eye, followed half an hour later by marked recovery in the upper field though the fixation area was still obscure. Ophthalmoscopically there was complete stasis in the superior retinal vessels; there was no evidence of spasm and no cherry red spot at the macula. Forcible intermittent pressure with a circular motion was performed for some minutes with a resulting increase in the area of field. The operator endeavoured to make the release of each push very sudden so as to give a forcible recoil. One hour after the attack the patient could see objects in all directions, though hazily, and eventually she regained 6/5 vision and a field nearly full except for a little contraction on the temporal side. Physical examination was negative and in particular there was no evidence of valvular disease. Beach has collected from the literature a series of 32 cases of recovery after obstruction of the central artery. Of these, 15 recoveries followed massage, 7 operation (iridectomy or paracentesis, sclerotomy), 2 the use of drugs (amyl nitrate and potassium iodide), 3 were spontaneous, and the remainder were treated by a combination of methods. The evidence from these cases shows that iridectomy or paracentesis is well worth while doing if massage is a failure, and von Pflugk had one remarkable case in which he obtained almost complete recovery after repeating his paracentesis for nearly three weeks (number of operations not stated). The inference is, that in these cases the nutrition of the retina is maintained by seepage of blood past the block in the vessel and that no case should be given up until treatment has been persevered in for a very considerable period.

F. A. WILLIAMSON-NOBLE.

(4) **Villard, H. and Dejean, Ch.—Thrombosis of the central artery of the retina. (Thrombose de l'artère centrale de la rétine.)** *Ann. d'Ocul*, CLXV, p. 241, 1928.

(4) **Villard and Dejean** describe clinical and histological findings in the case of a woman aged 71 years, who lost the vision of the right eye by thrombosis of the central artery of the retina and not by embolism. The presence of evidence of endocarditis favoured a diagnosis of embolism, but on the other hand the history was suggestive of thrombosis. The loss of vision was preceded by brief attacks of obscuration which increased in frequency and in severity until finally the eye became totally blind. At the time of examination of the patient there was vision = 1/50, optic disc very pale, retinal arteries very narrow, no macular oedema and no foveal red spot. Two months later acute glaucoma supervened with complete loss of vision in the affected eye. Enucleation was performed. Histological examination of serial sections showed a large organised mass in the central artery. The mass was closely adherent along a thinned roughened area of arterial wall where the endothelium was missing and the elastic tissue interrupted. The histological evidence was therefore incontrovertible, that thrombosis had occurred gradually, starting along a longitudinally placed area of ulceration of the intima.

HUMPHREY NEAME.

(5) **Barkan, Dr. Hans (San Francisco).—Air embolism of the retinal vessels.** *Arch. f. Ophthal.*, July, 1928.

(5) Before describing his own case **Barkan** reviews the literature of the subject and quotes a most interesting account by Beecher of ophthalmological observations made in a monkey which had air injected into its carotid artery. Almost immediately after injection, silvery bubbles and air columns were observed to shoot along the retinal arteries which in one or two seconds became completely filled with air. At the same time the papilla became pale and very soon after the entire fundus was seen to be traversed by innumerable very fine glittering lines which represented the capillary network of the retina. Barkan's own experiments were made with rabbits in which air was injected into the right ventricle. The immediate effect was to produce clonic convulsions and the appearance of bright silvery rod-like particles shooting along the retinal vessels, to be followed by " an almost volcanic eruption of air " which within a few seconds delineated the entire arterial tree as a brilliantly shining object. Numerous emboli were also seen in the choroidal vessels which became almost entirely white. The article concludes with an account of air embolism in a man following an operation on the lung. The fundus was not examined till some

months afterwards when it was found to be normal, though there was a scotoma in each field of vision due probably to partial destruction of the cells in the calcarine cortex.

F. A. WILLIAMSON-NOBLE.

(6) **Suganuma, Sadao (Tokyo).—On the pathogenesis of tuberculosis of the retinal vessels. (Ueber die Entstehungsweise der Gefässtuberkulose der Netzhaut.)** *Arch. f. Ophthal.*, Vol. CXVIII, 1927.

(6) It has been established that recurrent haemorrhages into the vitreous and retina, seen in adolescents, are almost exclusively due to a tuberculous periphlebitis in the retina. **Suganuma** shows that clinical, pathological and experimental investigations go to prove that this periphlebitis is in general a primary affection in the eye. His findings disprove the view, held by some writers, that this condition arises secondarily from a tuberculous lesion in the anterior portion of the globe by way of the lymph stream, but he admits that this may be a possible mode of origin in exceptional cases. The tubercle bacilli, according to the author, are carried to the eye by the circulation from tuberculous foci in other parts of the body, and in all probability pass into the perivascular lymph space from the post-capillary portion of the blood vessels, when they settle down in the most favourable sites, and form specific nodules round the vein.

THOS. SNOWBALL.

BOOK NOTICES

Manuel des Maladies de l'Oeil. Par CHARLES H. MAY, New York. Cinquième édition française. Pages 496, Figs. 374. Paris: Masson et Cie. 1929. Prix 55 fr.

This, the fifth French edition of May's Handbook, corresponds to the latest, *i.e.*, the twelfth, American edition, of 1927. In view of the numerous manuals and treatises on ophthalmology by French writers, the call for a fifth edition of this book is indicative of its wide acceptance by French students and others. This is no doubt partly due to the author's successful effort to combine brevity and clarity in his writing.

In arrangement this book does not differ noticeably from most small handbooks. It is essentially practical and little space is given to discussion of theories or knotty questions. Moreover, only a very

limited number of the great variety of operative procedures are described, but these are well and clearly done, and should suffice for the beginner. The section on Ocular Therapeutics, which deals with drugs and appliances and "general rules for operations on the eye," should prove very helpful to students and general practitioners. It is curious to find in an up-to-date book by an American author, the old notation of 20/20 etc., and still more curious to find this reproduced in an edition for French readers to whom measurements in feet must be purely historical.

The illustrations are numerous and generally good: a few of the uncoloured figures might be replaced by more modern drawings. The book is well printed on rather glossy paper; the coloured plates are well reproduced.

Grundriss der Augenheilkunde. By Prof. A. BRÜCKNER and Prof. W. MEISNER. 2nd Edition, Pp. XXV + 601 with 221 illustrations and 9 coloured plates. Leipzig: Georg. Thieme. 1929. Price, M.25.

This book, intended for the use of the student, makes a useful addition to the textbooks in existence. Its most valuable feature is the fact that it is essentially dual in nature, being a combination of a readable elementary textbook and a laconic guide to more advanced study. On matters of daily experience the book is no fuller than the smaller textbooks in English, such as the one by de Schweinitz, but the discussions on the rarer diseases and on subjects of theoretical interest, though slight, are sufficient to remind the student of the vast extent and ramifications of ophthalmology, a point not always appreciated by the non-expert.

The book is, of course, not devoid of faults. The subject of retinal arterio-sclerosis is dealt with in hardly more than a passing reference; diabetic retinitis is dismissed in a short paragraph, and, in common with other books, nystagmus is tabulated rather than discussed. Motor anomalies of the eye, a difficult and important subject, deserve more than nineteen pages, and the slit-lamp is certainly entitled to more than two paragraphs in small print.

But these defects do not detract from the distinct individuality of a book in which room is found for useful and interesting sections dealing with such subjects as the eye as a double organ, the biological significance of vision, the social problems of ophthalmology, and with the questions of racial hygiene and those of heredity in their relation to the speciality.

A pleasant feature of the book is its handsome appearance—a marked contrast to the first edition, which was published shortly after the war.

Annual Report of the Department of Health, Government of Palestine. 1927

The long and interesting Report of the Director, Col. G. W. Heron, C.B.E., D.S.O., on the activities of the Department of Health of the Government of Palestine, contains a section on the ophthalmic service, as usual.

The six Government ophthalmic clinics at Gaza, Ramleh, Nablus, Tulkarem, Acre and Beersheba, continued to function under the expert supervision of the Warden and surgeons of the Ophthalmic Hospital of the Order of St. John of Jerusalem. The following table gives a detailed summary of the work performed by the clinics. As compared with 1926, there was an increase of more than 2,000 new patients whose numbers reached 15,928. The increase is largely accounted for by the serious epidemic of acute conjunctivitis which occurred in the late summer. The total attendances—108,437—were not much greater than in 1926.

The following extracts from the Report for 1927 of the Warden of the Ophthalmic Hospital of St. John indicate the amount of work carried out by the hospital, the relation of the hospital to the Government ophthalmic clinics, and the seriousness with which acute conjunctivitis in Palestine must be regarded.

"The number of new patients seen during the year 1927 was 20,051, and the total consultations 85,564. The number of patients admitted to hospital was 1,391; and the number of operations performed 3,651. Classified according to religion of the new cases 12,998 were Moslems; 3,574 were Christians; and 3,479 were Jews. Of the total consultations 54,859 were Moslems; 14,307 were Christians; and 16,398 were Jews. The number of new patients is the largest yet recorded but the other figures show a slight decrease. The probability of this decrease has been mentioned in previous reports. On account of the activity of the ophthalmic clinics in different parts of the country, a large and increasing number of trichiasis cases are dealt with in their own districts. Further, these clinics sort out and send on to us the intra-ocular cases. In this way the in-patient department of the hospital deals with an increasing number of intra-ocular cases, and as these operations, all things considered, take longer to perform and the patients have to be retained longer in hospital, there is a corresponding fall in the number of operations performed and the number admitted to hospital. And this is as it should be if the country is to benefit to the maximum extent from the skilled staff and the well-equipped hospital of the Order. The year before the inauguration of the clinic scheme 66 per cent. of our operations were for the relief of trichiasis. In 1927 this had fallen to 53 per cent. and the intra-ocular operations had risen proportionately." Acute conjunctivitis,

although endemic or always present in the country, assumes epidemic form shortly after the onset of the hot weather and rages with ever-increasing virulence until the onset of the rains in November. The epidemic of 1927 was even more severe than that of 1926 (which was also a bad year). The acute cases numbered 4,291 and formed 21.4 per cent. of the new cases. During the early months of the year the cases averaged about 60 per month; in May they increased to 101; June, 273; July, 487; August, 1,115; September, 906; October, 639; November, 408; December, 194. The acute conjunctivitis was complicated with corneal ulceration in 752 cases. Of these 280 were perforating ulcers: a dreadful reinforcement to the kingdom of the blind, for the majority of these ulcers do not get to us until the damage is irreparable."

The measures taken for the prevention and early treatment of trachoma and acute conjunctivitis among school children are mentioned in the section of this Report, which deals with the School Medical Service.

Each year more than 80,000 new ophthalmic cases are treated at the various hospitals and clinics and more than 60,000 school children are kept under medical observation or treatment for their eyes.

Giza Memorial Ophthalmic Laboratory. Second Annual Report. Department of Public Health, Egypt.

The Giza Memorial Ophthalmic Laboratory has been luxuriously designed and equipped for research work, routine pathological examinations, and post-graduate education.

In a country where 90 per cent of the population either are suffering from trachoma, or show traces of the disease, it is natural that great interest is taken in the aetiology of this disease. It is interesting to note that almost immediately after the news of Noguchi's discovery of an organism, isolated from cases of American Indian trachoma, and shown to produce trachoma-like lesions in a certain type of monkey, Dr. Rowland Wilson and Dr. Haig, at the Laboratory, isolated an identical or similar organism from their abundant material in Egypt.

Various new methods of treating trachoma and its complications have very properly been tried in a scientific manner but without any important results.

A number of cases of primary glaucoma have been treated by intravenous injections of hypertonic saline solution, and of calcium chloride solution, also subconjunctival injections of glaucosan have been tried. In subacute cases from 30 to 55 c.c. of 30 per cent. saline solution were used. An hour after injection the corneae which had been steamy became clear, and the tension fell from about 60 to 30 by the Schiötz tonometer. In chronic cases, while

the tension fell after the injection, it began to rise again after 36 to 48 hours. In all cases operation was performed later. This method of treatment depends on osmosis while calcium chloride intravenously has an inhibitory effect on exudation and transudation. A 10 per cent. solution of calcium chloride to the amount of 10 c.c. was injected slowly into the median basilic vein. The blood pressure was not much altered by this dosage but almost immediately there occurred a great slowing of the pulse rate together with a sensation of heat all over the body and a feeling of apprehension. In the four cases selected for the treatment it required one injection on three to five successive days before the tension was reduced to normal, and another injection was usually required every three to seven days to keep the tension about normal. All these cases were finally submitted to operation.

Hamburger's glaucosan was used, also experimentally, in five cases of chronic glaucoma, it being considered unwise to use a solution containing adrenaline in acute cases, in the light of previous abundant experience of its effect in such cases in raising the tension still higher. The solution employed consisted of dextrorotatory adrenaline (1:500) and methyl-amino-aceto-pyrocatechin (1:100) in equal parts, 0.3 c.c. being injected in each case. The injection produces a powerful vaso-constriction and in doing so forces the blood out of the uvea. This is later followed by a hyperaemia similar to that which occurs in cyclitis and, with it, a reduction in the tension. The treatment was carried out in seven chronic cases. In no case, even with the addition of eserine instillations, did the tension remain at a normal level for more than 48 hours.

Among the routine work of the laboratory about 3,000 bacteriological and about 500 pathological examinations were made. Among the latter was a specimen of schistosomiasis (*Bilharzia*) of the conjunctiva, which has been described by Sobhy. This case is unique in ophthalmic literature. It occurred in a boy aged 8 years, and appeared as a small tumour firmly adherent to the upper tarsus. At the upper part of the limbus, and encroaching on the cornea there was an oval, flattened, vascular swelling of the bulbar conjunctiva showing several yellowish spots resembling miliary tubercles. Pathological examination showed ova of *schistosoma haematobium* present in the tissues in large numbers. Terminal spines were well seen in some of them. The eggs were surrounded by a zone of endothelial cells, fibroblasts, and many giant cells. Outside this was a zone of plasma cells. No eosinophiles were present. Ova with terminal spines were present in the urine. The boy was put on a course of tartar emetic lasting for three weeks. After six weeks the condition had practically disappeared.

Two courses of lectures on ophthalmology and ophthalmic pathology are held every year.

CORRESPONDENCE

PSEUDOGLAUCOMA

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIR,—Mr. Ernest Thomson, in his description of his interesting disc case reported in your March issue, refers to my case reported in the February number. He asks if there was any history of birth trauma in my case.

I have made enquiries and find that the birth was a natural one, the only abnormality being that the child was born "in a caul." The mother, who has had 11 children, states that instruments were never used in any of her confinements.

The only trauma known of in the case was the fall the mother had about seven months before the child was born.

Yours truly,

ALEX. MACRAE.

OBITUARY

JULES BADAL

WE note with regret the death of JULES BADAL, one of the doyens of ophthalmology in France. Born in 1840, Badal began his medical career in the army. He soon left this service, and joined the clinique of Giraud-Teulon in Paris, to devote himself to ophthalmology. In the war of 1870 he served in the army and for his conduct in the siege of Paris was awarded the Cross of the Legion of Honour. In 1878 the Faculty of Medicine of Bordeaux was founded, and Badal was given charge of the ophthalmic clinique. He continued his work there as Professor for 32 years. Badal was the author of a large number of papers on ophthalmic subjects and the inventor of a number of instruments and apparatus for use in clinical work; among these were his ophthalmometer and optometer, perhaps the earliest of their kind.

JAMES MOORES BALL

WE regret to record the death of Dr. JAMES MOORES BALL, of St. Louis, which took place just before Easter, from heart disease, at the age of 66 years.

Born in West Union, Iowa, he received his medical degree from the University of Iowa in 1884, and started in general practice, but since 1890 he confined himself entirely to ophthalmology, and was well known and respected in the States and in Europe. He was a member of the Ophthalmological Society of the United Kingdom.

He edited for a time the *Annals of Ophthalmology*, now merged in the *American Journal of Ophthalmology*, and was Professor of Ophthalmology at St. Louis. Only last month we noted his presentation to the St. Louis University of a valuable collection of books and prints relating to medical history.

His "Modern Ophthalmology" was a well-known text book. He was the author of a work on Vesalius, and only last year his "Sack 'em up Men," an account of the resurrectionists, was issued.

NOTES

Deaths

We regret to record the death on March 12, of Dr. J. MARTIN YOUNG, of Birmingham. He was taken ill suddenly in the street and was removed to the General Hospital, where he died. Qualifying from Aberdeen in 1889, he was ophthalmic surgeon to the Children's Hospital, Birmingham, and to the West Bromwich District Hospital. During the war he served as Captain R.A.M.C.(T.), attached to the 1st Southern General Hospital.

We also regret to record the recent deaths of Dr. TRUC, of Montpellier, Commander of the Legion d'honneur, at the age of 72 years, and of Professor FILIPPO SPECIALE CIRINCIONE, Junior, Director of the Ophthalmological Clinic at Turin, aged 47 years.

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The Gifford Edmonds Prize in Ophthalmology

THIS prize has been awarded to Mr. V. M. Métivier, of the Ophthalmic Department of Edinburgh Royal Infirmary, for his Essay on The Causation and Differential Diagnosis of Proptosis. This prize, founded by Miss Sophie Edmonds in memory of her brother who

fell at Magersfontein, is valued at £100 and is awarded every second year.

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St. Margaret's Hospital for Ophthalmia Neonatorum

THE annual returns for St. Margaret's Hospital for ophthalmia neonatorum for the years 1927 and 1928 are as follows:—

No. of children admitted		No. of mothers admitted		No. of children discharged with both eyes blind		No. of children discharged with single eye blind		No. of eyes with impaired vision	
1927	1928	1927	1928	1927	1928	1927	1928	1927	1928
211	204	143	138	Nil	Nil	1	3	20	23 (6 both eyes 11 single eye)

* * *

D.O.M.S., London

THE following were successful at the recent examination for the Diploma in Ophthalmic Medicine and Surgery;—S. Annaswami, Y. P. Bedi, G. R. Bhatia, M. D. Bower, G. A. Bradshaw, C. S. Colvin, H. V. Coverdale, H. N. Dewan, Jean M. Dollar, J. B. Dhaubhoora, E. H. W. Elkington, W. D. Hamilton, M. Husnain, Audrey H. Henson, S. J. A. Laubscher, A. A. El M. Magraby, Sigrid L. S. Pearson, F. E. Preston, Gertrude E. Pugmire, B. W. Rycroft, A. E. M. Soliman, A. D. C. Stoute, Annie V. Taylor, Marjorie P. Taylor, and G. Vaishnavi.

FUTURE ARRANGEMENTS

1929

May 13-16.—Société française d'Ophtalmologie. (Annual Congress), Paris.

June 14.—Section of Ophthalmology, Royal Society of Medicine. (Annual Meeting).

July 4-5.—Oxford Ophthalmological Congress, at Oxford.

September 5-13.—**International Congress of Ophthalmology at Amsterdam.**

CONTEMPORARY OPHTHALMIC LITERATURE

Archives of Ophthalmology. *February, 1929.*

SMITH. The nutrition of the lens and of the vitreous.
WILMER. Cyst of the uveal layer of the disc at the pupillary margin in a case of posterior synechiae with secondary glaucoma.
CUSHING and EISENHARDT. Meningiomas arising from the tuberculum sellae with the syndrome of primary optic atrophy and bitemporal field defects combined with a normal sella Turcica in a middle-aged person. *(Concluded).*
LING. Interstitial keratitis with unusually marked chorioretinitis. Pathological-anatomic examination of a case.
HOLLOWEG and VERHOEFF. Disc-like degeneration of the macula, with microscopic report of a tumour-like mass in the macular region.
KRONFELD. The regeneration of the aqueous humour.

American Journal of Ophthalmology. *March, 1929.*

LOWELL. Measured correction of a squint by combined technique.
KEY. A case of trichinosis with exophthalmos.
O'ROURKE. The optical correction of conical cornea with the contact glass.
KNIGHTON. Hyperopic under-correction due to retinoscopy.
EVANS. A scotoma associated with strabismus.

Annales d'Oculistique. *February, 1929.*

DUBOIS. Traumatic retinal detachment and its operative treatment.
TERSON. Treatment of choice for spontaneous entropion of the lower lid, with pathological notes.
VALIÈRE-VIALEX. Treatment of choice for so called spasmodic entropion. Operative procedure.
BRUCKNER. Contribution to our knowledge of the immunity of the eye; the influence of ocular hypotension on the quantity of haemolysins in the cornea (in the aqueous humour and the vitreous) in normal and immune rabbits.
BIRNBAUM. Conjunctivitis and its treatment by "Targésine."

Archives d'Ophtalmologie. *February. 1929.*

TERRIEN. Remarks on tumours of the iris.
GABRIÉLIDÈS. Galien's optotype.
PARLANGE. Ocular sequelae of poison gas.

La Clinique Ophtalmologique. *December, 1928.*

PARFONRY. Surgical treatment of paralytic strabismus *(continuation and end).*
MORISOT. Ionisation in ocular therapy *(continuation and end).*

Société d'Ophtalmologie de Paris. *January, 1929.*

WORMS and LACAZE. Persistence of the hyaloid artery and the canal of Cloquet.
POLACK. Concerning the ocular examination of emigrants.
PLICQUE. Experimental and clinical study of the action of the thyroid on the ocular tension.

DE ST. MARTIN. Oculo-motor complications of ophthalmic herpes. Trophic disturbance of the iris; herpes and chicken pox.
HARTMANN. Radiological aspect of cerebral tumour near the sphenoidal fissure.
MAGITOT. Four new observations on intra-cranial hypertension without ophthalmoscopic signs.
On epiphora without stenosis.
LAMACHE and DUBAR. Transitory papilloedema.
DUPUY-DUTEMPS. On the pathogenesis of papilloedema.

Klinische Monatsblätter für Augenheilkunde. *February, 1929.*

DE VRIES. X-ray treatment of cancer of the eyelid.
DEN HOED, STOEL and DE VRIES. Investigations into the means of protection of the eye in ray treatment of the lids.
SELENKOWSKY and MALISCHEFF. On the treatment of trachoma with radium emanation.
ROHRSCHNEIDER. A new prosthesis for the protection of the eye from X-rays and radium rays.
RAUBITSCHEK. Observations on skiascopy with cylinders.
KOYANAGI. Dysacusis, alopecia and poliosis in severe non-traumatic uveitis.
TRIEBENSTEIN. On the question of Mooren's ulcer.
SAEGER. On the control of corneal vascularisation by means of the caustic pencil with remarks on filariasis of the eye.
V. GERNET. Criticism of Kreiker's "implantation of oral mucous membrane in the intermarginal strip."
WIEGMANN. Case of psammoma of the orbit.
HAMBURGER. In support of Prof. W. Stock's paper on the treatment of glaucoma.
ARRUGA. An alteration in the shape of the trephine to facilitate the boring of the bone in Toti's operation.
WECKERT. On combating amblyopia from squint.
KNAPP. Note on the question of the after effects of the removal of the lacrimal gland.
AXENFELD. Observations on the changes accompanying extirpation of the palpebral portion of the lacrimal gland.

March, 1929.

SEEFELDER. Is enucleation of the eye or exenteration of the orbit preferable in sarcoma of the choroid.
DEUTSCH. On the alteration of irregular astigmatism and high ametropia with contact glasses.
MEYER. Research into the toxicity of trichlorethylene, particularly on the eye.
KOYANAGI. On the overhanging retina in the disc in glaucoma.
ISHIKAWA. Observations on progressive sclero-perikeratitis (v. Szili).
HORNIKER. On the question of albuminuric retinitis.
KAMINSKAJA-PAWLOWA. Changes in the iris in tabes.
GASTEIGER. Treatment of diseases of the cornea with ultra-violet light.
SABATZKY. Increased occurrence of optic neuritis as a complication of influenza.
THIES. Severe burning.
MEYER-WALDECK. On treatment of spastic entropion.
FLASCHENTRÄGER. Iris diaphragm in weak sight.
SHIMKIN. Three cases of spastic entropion after complete tarsectomy of the upper lid.
BETSCH. On the human refraction curve.
KRAUPA. On senile destruction of the lens capsule.
BRANA. Remarks on D. Mikaeljan, Kruglow, and Tarnopolsky's papers on the connection between constitution and trachoma, from the Trachoma Clinic in Kasan.
RÖNNE. A concave loupe as optical assistance in retinitis pigmentosa.

Zeitschrift für Augenheilkunde. *February, 1929.*

FUCHS. On the structures of the cornea and the marginal ring of Schwalbe.
ACHERMANN. Work on the heredity of v. Recklinghausen's disease.
KOFLER. On minute anatomical details and variations in the lacrimal fossa of the frontal bone.
BISTIS. Affection of the eye in dengue fever.

March, 1929.

HORNE and KOGERER. On further investigation of cases of tabetic optic atrophy treated by air injection.
HAMANN. Aneurysm of the internal carotid.
REIGER. Stargardt's prophylaxis of ulcus serpens with optochin.
SPANUTH. Thirty years of non-reflecting ophthalmoscopes.

Annali di Ottalmologia e Clinica Oculistica. *February, 1929.*

LO CASCIO. Researches on the dehydrogenating processes in the retina.
TESSIER. On functional disturbances of the iris and ciliary body.
SABA. Ocular changes in post-malarial pernicious anaemia.
ANTONIBON. Researches on the absorptive power of the cornea.
REGANATI. On the cure of trachoma with chaulmoogra oil.

Lettura Oftalmologica. *October, 1928.*

SANTORI. Contribution to the aetiology and pathogenesis of unilateral anophthalmia, and histological report on bilateral anophthalmia.
LA FERLA. Sensori-motor ophthalmoplegia of sinus origin (syndrome of orbital apex).

November, 1928.

NICHEL. Clinical and experimental contribution to the study of Parinaud's conjunctivitis.

December, 1928.

DENTI. Actinomycosis of the conjunctiva.
ADINOLFI. A case of serious ocular tuberculosis following removal of a nasal polyp.
SALVATI. On serological immunity in trachoma.
MAZZOLA. Tracholisina in the cure of trachoma.

Bollettino d'Oculistica. *January, 1929.*

ALAJMO. Biomicroscopy in ocular trauma (medico-legal aspect).
ACCARDI. Intra-ocular tension in strabismus.
SANTORI. On a special form of iris atrophy in dislocation of the lens.
NICOLATO. Muscular stripping (Duverger's operation) in the cure of strabismus.
AUBARET. Reduction of the iris in cataract operation with the double needle.
KRETZ. The relation between astigmatism and the myopic crescent.
DENTI. Keratitis in quinine poisoning.

February, 1929.

TRISTAINO. The general condition, especially the renal efficiency and the oculo-cardiac reflex in cases of detached retina.
ALAJMO. Biomicroscopy in ocular trauma (continued).
MEZZATESTA. Contribution to the treatment of corneal tubercle.
SANTORI. Large congenital naevus of the iris hanging in the anterior chamber